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THE BASIC MECHANISMS OF PSYCHOSOMATIC DISORDER.

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FROM earliest times correlations have been made between emotional states, physical habitus and somatic disorder, but during the last century these became largely disregarded because of the growth of medicine into a genuine natural science based on the enormously successful application of the principles of physics and chemistry.

It is one of the paradoxes of historical development that the greater the merits of a method or principle, the greater will be its effect in retarding subsequent progress, and it is only recently that a proper study has been made of the psychological aspects of physical disease. As a result it has been reserved for the psychiatrist to introduce a new synthetic approach to medicine, and it is general medicine that has begun to adopt this orientation rather than the reverse.

This orientation is called the psychosomatic point of view. It is one in which there is a coordinated use of somatic and psychological methods and concepts in the study and treatment of disease. The psychosomatic field of reference implies a continuum. This is not homogeneous, but has recognizable nodes of organization, which include soma, psyche, group, society and culture, all in the four dimensions of space and time. These nodes or foci are in reciprocal relationship, but in any investigation one tends to appear to be isolated because it is rarely possible to investigate or master more than a part of a larger problem.

The word psychosomatic unfortunately implies a dichotomy of mind and body, but it is too firmly established to be dislodged even if a more suitable one were found. It appeared first in Germany as *psychosomatische*, being used by Heinroth in 1818. The anglicized form of the adjective appears to have been introduced by Flanders Dunbar in 1934 (Dunbar, 1948).

In using the word one must consider whether the allusion is to (*a*) the functional manifestations of emotion, for example, tachycardia in anxiety states; (*b*) symbolic dysfunction of emotional origin, as, for instance, paralysis in conversion hysteria; (*c*) structural disease in which emotional disturbance is a possible cause, such as ulcerative colitis; or (*d*) sickness in which the whole person —mind and body—is disturbed, as in any illness.

The term psychosomatic disease as a diagnostic or classificatory concept was proposed by Halliday (1943) on the assumption that in a certain group of organic diseases the outstanding ætiological factor was psycho-

logical. Obvious examples of this are duodenal ulcer and ulcerative colitis. To these might be added certain well-defined syndromes in which there is no structural change, such as migraine and uncomplicated asthma. Organic changes, as the contractures seen in a chronic hysterical paralysis or those of malnutrition resulting from a psychosis, though obviously psychosomatic, fall into a different category. Manifestations as simple tachycardia or functional palsy could well be termed disorders rather than diseases.

Mechanisms.

For emotional changes to produce alterations in the function, and possibly in the structure, of a tissue, a physiological mechanism is required to link the cortex and the target cells. In recent years a considerable amount of knowledge has been gained regarding the nature of this mechanism and since homœostasis is maintained by the nervous and vascular systems, its effects are believed to be mediated through the nervous and endocrine channels.

Before these links are discussed, reference must be made to the types of emotional disturbance associated with these disorders. They are the common neuroses.

Types of Emotional Disturbances.

The first point to make in this connexion is that for each emotional state, for example, rage or grief, there is a specific syndrome of physical changes such as are seen in alterations of the blood pressure, the respiratory rate, or the colour of the skin, or in the "expressive movements". These changes are not related to conscious or unconscious goals, but are the result of the discharge of emotional tension in a complex stereotyped pattern, common at least to the species. With acute emotions these changes are transitory; in chronic states prolonged bodily disturbances may develop.

The first chronic state to be studied, however, is that for which Freud introduced his concept of conversion hysteria. Here the changes are in voluntary movements or in peripheral sensory perception, that is, in organs that subserve the external relations of the individual—the organs of the ego. The motivating impulse or goal is an unconscious one, and the symptoms are not stereotyped reactions but individual creations expressive of the particular repressed psychological content.

A fundamentally different group of chronic disturbances is that involving the internal organs. Some workers believed that all psychological disturbances of the body were hysterical conversions, and for them the elevation of the blood pressure in rage would have a symbolic meaning. Dunbar (1940), however, showed that these disturbances —many of which the general physician would call genuine somatic illness—need not be symbolic, and Freud taught that, as the involved organs and their functions are hidden from direct knowledge, that channel of expression could not be used to explain the visceral neuroses. Alexander (1952) also denies that the changes in these neuroses are an attempt to express an emotion, and considers that they are the physiological response to chronic emotional states—conscious or unconscious. An elevation of the blood pressure does not relieve rage, but is a component of the stereotyped rage phenomenon.

The orifices mark the dividing line between the external and the "internal" environments. Their functional activities are initiated by the voluntary nerves and carried on by the autonomic. They can therefore be involved in both types of disorder—the hysterias on the one hand and the visceral neuroses on the other. Examples are seen in vomiting and disturbances of micturition of nervous origin. The skin, too, has a dual nerve supply and can fill both roles.

Up to this stage the mechanisms discussed have been for functional disorders. Many now believe that prolonged functional disturbance may lead to organic change. This has been indicated especially in the psychosomatic studies on peptic ulceration and ulcerative colitis. Wolf and Wolff (1943), for example, showed that the stomach reacted to certain emotional states with hyperæmia, hypermotility and hypersecretion. When these were present, the symptoms were as in peptic ulceration; and actual ulceration was occasionally seen in these periods. If this be so and altered function can cause organic change, it reverses a usual concept that disturbed function is the result of altered structure. All might not agree on the mechanisms involved, but I do not think that this matters practically at this juncture, provided one can establish a relationship —specific if possible—between certain emotional states and certain physical diseases.

Up to this point the discussion can be summarized thus:

1. All healthy and sick functions are psychosomatic.

2. Emotions are always associated with autonomic action patterns.

3. For specific emotions there are appropriate patterns.

4. Suppressed emotions cause chronic tension, which intensifies and prolongs the changes.

5. Excessive organ innervations leads to functional and eventually to structural change.

The Role of the Autonomic and Peripheral Nerves.

An all-over function of the nervous system is to maintain the organism in a constant state—that is, in homœostasis. The peripheral nerves regulate the relations with the external environment and the autonomic nerves those with the internal. Of the latter, the sympathetic nerves adjust the internal economy to external activities, and particularly to emergency situations, that is, to fight and flight. Their function is katabolic. The parasympathetic nerves, on the other hand, are concerned with protection, conservation and building-up—with anabolism—and in general their functions represent a withdrawal from external activities.

The emotional disturbances of vegetative function can be divided into two main groups—those related to the everyday function of preparation for fight or flight, and those related to withdrawal from outwardly directed activity. Disturbances of the first group are caused by inhibition, repression or blocking of self-assertive or hostile impulses, so that the corresponding behaviour (fight or flight) is never consummated, although the organism is in a state of chronic preparedness. Diseases believed to be the result of this are hypertension, migraine, *diabetes mellitus*, rheumatoid arthritis and thyrotoxicosis. In the other group—the withdrawal one—are many diseases of the alimentary canal, such as obesity, duodenal ulcer, colitis and constipation, and also asthma and fatigue states due to mild hypoglycæmia. It would seem obvious that in the first of these there will be a sympathetic and in the second a parasympathetic preponderance; but this is a simplification, because at all times both divisions are working together, and any disturbance of equilibrium will initiate a compensatory reaction tending to restore homœostasis, and this reaction may then appear to be the primary one. The longer the disturbance prevails, the more complex will be this mixing.

The peripheral motor nerves are involved in such conditions as tension states, in which muscular rigidity is the result of anxiety, and both motor and sensory disturbances are seen in hysteria.

The Role of the Endocrine Glands.

Apart from the nervous pathways that have been described, emotional states may affect the body by means of the endocrine glands and their secretions. In general, rapid adaptations are effected by the nervous mechanisms and slower and more enduring ones through the endocrine system. Between these two, homœostasis is maintained. Both are controlled by the hypothalamus and its connexions.

The adenohypophysis is linked with the hypothalamus by the hypophyseal portal system. There is no known nervous connexion. It is influenced also by the inhibitory effect of excessive secretion of its target glands and

through sympathetic activity causing the peripheral liberation of adrenaline, which results in the secretion of ACTH and probably other of its tropic hormones. The neurohypophysis seems of less importance in these states.

It will now be seen that the organism may prepare to meet a stressful situation in three ways: (i) by activation of a system comprising the sympathetic nerves, the adrenal medulla, the anterior pituitary lobe and the adrenal cortex; (ii) by using the parasympathetic type of response; or (iii) by retreat by consciously seeking aid.

Mention of ACTH draws attention to cortisone, and it is noteworthy that most of the diseases that are relieved by this steroid have been regarded as psychosomatic ones (Figure I).

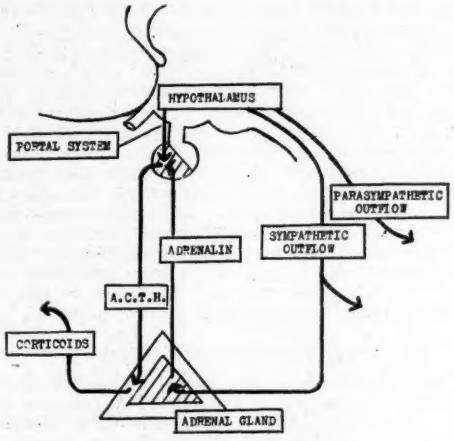

FIGURE I.
The neuro-endocrine mechanism.

Basic Conflicts.

Nervous and endocrine responses to emotional disturbance have been discussed. The emotional changes are believed to be the result of psychological conflicts. Some relevant views on these are now presented.

Dunbar (1943) had concluded that the basic conflict in her patients was around the tendency to fight or to take flight. Alexander (1952), whose early work was largely with gastro-intestinal diseases, at first believed that it was around the desire to take in and retain on the one hand and to give or expel on the other, these wishes being seen as the psychic expression of the basic functions of the alimentary tract and, be it noted, of its embryonic appendages the respiratory and urinary tracts. From later observations he came to the conclusion that the conflict was much more fundamental, and he employed a circular concept (Figure II). Starting with the universal need for dependency, he postulated that, if frustration occurred from the blocking of direct outlets, protest and overcompensatory activity would result and be manifested, on the one hand by parasympathetic overactivity with its protective and conservative functions and on the other by increased activity of infantile regressive trends. Should these efforts meet with further frustrations, the dammed up energy would overflow, leading both to increased sympathetic activity and to greater competitive aggressiveness. This exaggerated response might prove equally unacceptable to society and lead to further frustrations, until ever-increasing anxiety and guilt might bring about a regression to the original point of departure—the original state of dependency.

Organ Specificity.

The problem of specificity of reaction through sympathetic or parasympathetic channels has been discussed, the next question to consider is that of organ specificity. Why, for example, does one person react through the bronchial tree, another through the bowel and yet another through the skin?

In the field of organic pathology the focus was first on organs, then on tissues and, with Virchow, on cells. Today the trend is to regard a disease as a state affecting the whole body in which one or other organ is involved with special emphasis. Therefore, to approach the problem of specificity in psychosomatic medicine by reverting to the concept of organ disease is a regression. The fact remains, however, that here, as in organic medicine, certain organs do appear to be involved more frequently than others in response to specific stresses, and also that certain persons may act characteristically and in an individual manner to general non-specific ones. Thus Malmo (1954) and others have shown that when such functions as pulse or respiratory rate, sweating and muscular tension are measured during the application of non-specific stimuli, as from electric shocks, painful pressure, noise or mathematical problems, each individual will react characteristically. For example, those prone to headaches show a generalized increase in muscle tension and those prone to palpitations an increase in pulse rate.

In organic medicine we know that any sign or symptom may be caused in a variety of ways, and further, that a collection of these, that is an illness, may be regarded as representing the resultant of the action of many forces. Thus pyrexia may be due, among other things, to an infection; and if this be tuberculous, it is then related to every part of the constellation of forces, physical, sociological and epidemiological, required to bring about active tuberculosis in an individual case. If the same propositions be stated for a psychiatric illness, then the network

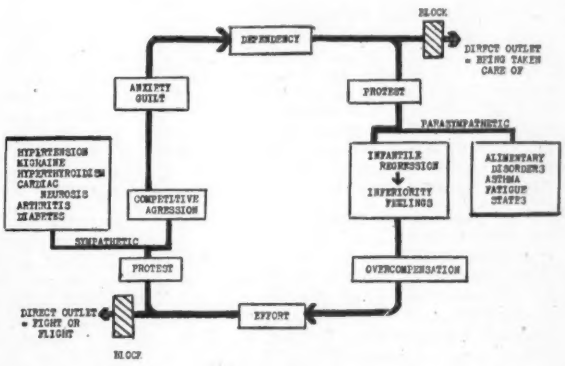

FIGURE II.
Alexander's circular concept.

of events in which a psychosomatic disorder is a point is much more intricate than that for either a purely organic or a purely psychological one—if such occur. Some of these will be related to the problem of organ specificity.

This approach seems more reasonable than those in which such terms as "selection" or "choice" of an organ are used, except possibly in hysterical reactions involving symbolic mechanisms, and even there the point of view could be taken that the organ or tissue is vulnerable—not chosen.

The following are of psychological importance in this respect:

1. Emotional associations, conscious or unconscious, with different organs, tissues or functions. (*a*) Organic illness or injury in childhood will produce far-reaching results, which will depend on such factors as the site and severity, residual deformity and the conduct of those caring for the patient. An illness later on also may have the effect of secondarily distorting the functions of the organs con-

cerned. For example, when a patient has a gastrostomy, this must come to occupy a central place in his mind and must thus distort the normal attitude towards the stomach; so that the mutilated organ may become the primary one for expression of everything the patient thinks or feels. We have no right therefore to assume that the responses of a gastrostomy patient will represent the gastric functions of an intact human being without exaggeration, distortion, or both. (*b*) Vicarious emotional experiences when related to illness or injury in one to whom the subject is emotionally attached need no comment. (*c*) Freud has described the passage of the infant through oral, anal and genital stages. In neurotic states or in times of stress when the subject is unable to handle his problems in an adult manner, he tends to fall back to one of these earlier stages. In peptic ulceration and obesity it is to the oral stage, in colitis to the anal. (*d*) Another aspect of these associations is that of the theory of organ language, which presumes the involvement of an organ on the basis of a symbolic meaning for its structure or function. Diarrhœa may mean giving, expelling or soiling, the vomiting of pregnancy may mean getting rid of the fœtus, and skin diseases may be stigmata. Bound up with this are the common expressions such as "heart rending", "can't stomach or swallow it", "getting it off your chest" and the like. The different shades of meaning between, say, "he gets under my skin" and "he gives me a headache" are definite and are often most appropriate to the situations in which either skin disorders or headaches occur. The common people seem to be intuitively aware of their deeper significance. "Gesture language", as of actors, is an extension of the above.

2. The emotional climate of the family, group or culture. This may be most important. Reference to it is made below.

3. The question of diatheses or an inherent susceptibility. This has been advanced by Adler in his concept of organ inferiority, according to which in any neurosis compensation occurs for a constitutionally inferior organ system and thereby localizes the symptoms. Associations have also been made between habitus, temperament and disease. Sheldon's viscerotonic endomorph, for example, in his unregenerate state with his love of luxury and ritual eating, being prone to obesity and, according to Gertler *et alii* (1951), to coronary artery disease.

The position is complicated further by the fact that significant psychological influences, such as anxiety, repressed hostile and erotic impulses, guilt feelings and the like, are all present in all these disorders; so that it is not their presence that is specific, but the dynamic configuration in which they appear. Alexander (1952) has likened this specificity to that found in organic chemistry, where the constituents are few and the same, but are combined in a great variety of ways to give highly specific substances. Moreover, the biological maturation theory states that in early life the organism is plastic, but that later on the patterns of response become more rigid. Those who hold this theory stand half way between the dynamists of the psychoanalytical school and those who believe that all behaviour is the result of conditioned reflexes.

It will be seen, therefore, that the organ affected and the way in which it is disturbed will depend upon many factors, some of which are essentially accidental, in the final path to the particular symptom.

Personality and Disease.

So much for the emotional background to the different diseases. Can this now be applied to personality and disease? Are those with certain types of personality predisposed to certain diseases? The concept of a diathesis has already been referred to. It is an old one and was seen, for example, in the reference to the melancholy bilious subject. Most observations, however, were more or less anecdotal until Dunbar (1943) applied modern methods of study to this field. She made very full examinations of a large number of patients and by picking out common features formed her "personality profiles". She advanced the theory that each group had developed their type of mental machine as a result of faulty adjustment during infancy, and that each abnormal mental machine would correspond to a specific personality profile. Thus her coronary disease patient was a consistently striving person of great control and persistence—a long-term planner—and her fracture-prone patients were impulsive, unsystematic persons living for the present, often showing ill-controlled hostility to those in authority, and with guilt feelings and a tendency towards self-punishment and failure. She did not use psychoanalytical techniques, and this led to the criticism that her profiles only evaluate superficial aspects of personality.

The one danger of such profiles is their attractiveness. They so obviously apply to some patients that one forgets the others. Yet, for all that, there is much truth in many of them, and I think that a reason for this can be shown.

Alexander (1952), whose approach is psychoanalytical, believes that psychosomatic disorders are associated with specific emotional states rather than with specific personality patterns. Thus chronically sustained hostile impulses can be correlated with chronic hypertension and dependent help-seeking ones with hyperchlorhydria, and these emotional states may occur in a great number of different personalities. He admits that the "go-getter" type who represses and overcompensates is commonly found among peptic ulcer patients, but believes that this is related to our culture, in which great emphasis is placed upon independence and personal accomplishment. In other cultures other defences against the same emotional conflict would be found. This may well be a key to the understanding of many psychosomatic relationships.

The Neuroanatomical Frame.

In any disease only the cause is pathological; the remaining features, that is, the body's reaction, are physiological. The manifestations of a particular disease are the result of reactions of the particular tissue to the particular stress. Some aspects of the stresses have been discussed, as have also some of the ultimate disorders that are produced through the action of the autonomic and endocrine systems, but the links, as it were, between the mind and these remain for consideration. What then, is known of the neuroanatomical basis of behaviour?

It is now known that the archipallum—the old smell brain—has functions far beyond that of smell. To understand the modern conception of its function its comparative anatomy must be reviewed.

In primitive fishes the olfactory apparatus and the hypothalamus are so closely allied as to be practically indistinguishable. A step further on in the reptiles, the hippocampus, amygdala and olfactory apparatus occupy most of the forebrain. MacLean (1954) calls this the "neural chassis". It provides a mechanism for giving stereotyped reflex reactions to messages received from the external and internal environments. This "chassis" lacks a driver to give it intelligent direction, and later this is provided by the neopallium. In man it comprises (*a*) the archicortex, which is associated with the medial olfactory tract, the hippocampus and the dentate gyrus; (*b*) the palæocortex, of which the lateral olfactory tract ending in the pyriform lobe is a part; (*c*) the mesocortex, from which is derived the cingulate gyrus (Figure III).

These three form the great limbic lobe of Broca. It occupies the same position in all species, and in all has a relatively simple neural structure compared with the neocortex. In 1937 Papez, a comparative neurologist, called attention to this forgotten cortex and was the first to suggest that it constituted an "harmonious mechanism which might elaborate the functions of central emotion, as well as participate in emotional expression". This is the visceral brain. It has strong connexions with the hypothalamus and is well placed to receive and relate all forms of internal and external experience. Its simple cytoarchitecture has suggested that here experience may be interpreted in terms of feeling rather than of knowing or

of abstract or intellectual thought. The crudity of this analysing mechanism has been used to explain the confusion between internal and external perceptions observed in psychosomatic conditions, in which symbolical meanings may be given to organs or their functions, as when vomiting may mean rejection.

It has been shown by experiment and by studies of psychomotor epilepsy that this region is reciprocally connected with adjacent parts of the neopallium, such as the fronto-temporal region, which includes the posterior orbital, the anterior insular and the polar temporal areas. These discharge into the rostral hippocampus and into the amygdaloid nucleus. It has been long known that the pre-motor cortex influences various autonomic functions in a circumscribed or spot-to-spot relationship, but recent work indicates that the deeper areas of the temporal lobe and their connexions represent complex ones in a more organized manner. MacLean believes that these are especially related to oral activities pertaining to the vocalization, attack and defence involved in obtaining food. There are also present cardiac and respiratory connexions. The fact that the cerebrum is an outgrowth of the olfactory brain is evidence of the fundamental role of the sense of smell in obtaining food, in warning of enemies and particularly in sexual function. The medial olfactory tract leads to correlation

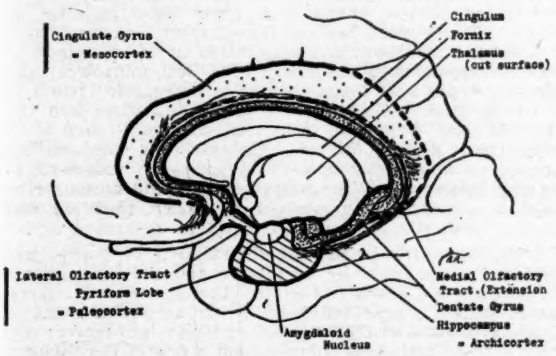

FIGURE III.
The limbic lobe.

centres of smell, taste and sensation from the mouth and viscera. MacLean has suggested that the amygdala may be regarded as an "olfactory" thalamus, serving here as does the optic thalamus with other sensory pathways.

On the other hand, it has been similarly shown that the region is associated with such emotions as rage and fear, and with changes in affective and sexual behaviour. Further, emotional tone as a whole is believed to be maintained by the passage of impulses to and fro, through closed circuits linking this fronto-temporal region with the hypothalamus, the thalamus, the reticular substance and the cingulum (Langworthy, 1955). Two well established ones are shown (Figure IV).

From these studies an understanding is gained of the kind of mechanism that provides the raw material for subjective emotion and relates this to the soma. Here presumably is a primitive part of the brain that expresses experience as feeling and determines whether or not impulses are released to the hypothalamus.

Phylogenetically it was not until the development of the supragranular layers of the neopallium associated with senses other than that of smell that it became possible to deal with a concept intellectually or in terms other than of emotional feeling. If emotional feelings are thus repressed or disregarded, there may be an increase in emotional tension manifesting itself as a disorder of behaviour, of function or of structure—that is, as a psychosis, a neurosis, or a psychosomatic illness.

Summary.

1. With the relatively recent growth of medicine into a natural science, earlier correlations between emotional states, physical habitus and somatic disorder largely became disregarded. These are now being reviewed; and coordinated somatic and psychological concepts and methods are being used in the study and treatment of functional and organic disease. From this point of view all healthy and sick functions are psychosomatic.

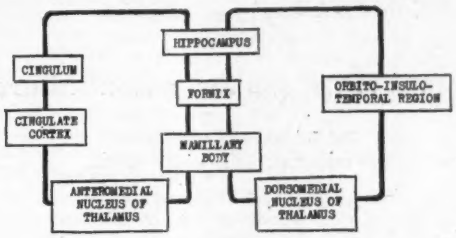

FIGURE IV.
Closed circuits subserving emotional tone.

2. Alterations in the function or structure of a tissue as a result of emotional changes must be by way of physiological mechanisms. These are believed to be mediated through the nervous and endocrine systems.

3. Specific emotions are always associated with appropriate autonomic action patterns. If the emotion is suppressed or disregarded, these are intensified and prolonged and give rise to functional and possibly structural change. The emotional changes involved are those seen in the common neuroses. These arise from basic conflicts, which may be met aggressively by activation of a sympathetic-adrenal system, passively by a parasympathetic type of reaction or by retreat by seeking aid.

4. Any individual will react characteristically to a stressful situation. His type of response, be it with functional or with structural change, is envisaged as representing the resultant of the action of many forces. These include conscious and unconscious emotional associations with the different tissues, organs and functions, the emotional climate of the family, group or culture, and diathesis or inherent susceptibility.

5. Each psychosomatic disorder appears to be related to a specific emotional state, rather than to a specific personality pattern. The latter association, however, is frequently found, and this may be because it represents an appropriate reaction in the particular cultural environment. In a different culture the disorder and its related emotional state could more frequently be associated with another pattern of superficial behaviour.

6. Emotional responses are now believed to have their origin in the archipallium. This region has strong connexions with the hypothalamus, in which the activity of both autonomic and endocrine systems is initiated. Thus a mechanism is provided by which subjective emotion may be related to the soma. If emotional states are repressed by neopallial activity, there may be an increase in emotional tension manifesting itself as a disorder of behaviour, function or structure.

References.

ALEXANDER, F. (1952), "Psychosomatic Medicine", Allen and Unwin, London.

DUNBAR, H. F. (1940), "Emotions and Bodily Changes", Columbia University Press (New York).

DUNBAR, H. F. (1943), "Psychosomatic Diagnosis", Paul B. Hoeber, Inc., New York.

DUNBAR, H. F. (1948), "Synopsis of Psychosomatic Diagnosis and Treatment", The C. V. Mosby Company, St. Louis.

GERTLER, M., GARN, S., and WHITE, P. (1951), "Young Candidates for Coronary Heart Disease", *J.A.M.A.*, 147: 621.

HALLIDAY, J. L. (1943), "Concept of a Psychosomatic Affection", *Lancet*, 2: 692.

HEINROTH, J. C. (1818), quoted by Margetts, E. L., in "Recent Developments in Psychosomatic Medicine", edited by Wittkower, E. D., and Cleghorn, R. A. (1954), Sir Isaac Pitman and Sons, London.

LANGWORTHY, O. R. (1955), "Newer Concepts of the Central Control of Emotions: A Review", *Am. J. Psychiat.*, 3: 481.

MALMO, R. B. (1954), in "Recent Developments in Psychosomatic Medicine", *loc cit.*

MACLEAN, P. D. (1954), in "Recent Developments in Psychosomatic Medicine", *loc. cit.*

PAPEZ, J. W. (1937), "A Proposed Mechanism of Emotion", *Arch. Neurol. & Psychiat.*, 38: 725.

WOLF, S., and WOLFF, H. G. (1943), "Human Gastric Function", Oxford University Press, New York.

QUAKER PHYSICIANS AND NONCONFORMITY.[1]

By K. MACARTHUR BROWN,
Parramatta, New South Wales.

Myself when young did eagerly frequent
Doctor and Saint, and heard great argument
About it and about; but evermore
Came out by the same door wherein I went.
Omar Khayyám.

LIKE their ancient predecessors in the land of Shinar, many Arabian scholars of the tenth century were still hopeful of being able to solve the riddle of life. However, in the long and laborious search for ultimate reality they were decidedly in advance of the Christian people of Western Europe, for the studies of the Arabian scholars were enhanced by their knowledge of the cultural achievements of Greece and Rome. First and foremost among the diverse philosophical speculations which continued to exercise the minds of Arabian doctors was the pressing need to effect a reconciliation between knowledge and religious belief. No such wanton and imprudent ambition could produce an equal response in the thoughts of learned schoolmen throughout mediæval Europe, whose ideas were stifled by their uncompromising and unquestioning attitude to human reasoning.

Nevertheless, the small principalities and powers of Europe were singularly fortunate in certain respects. Apart from an occasional outburst of excessive nationalism, the people were privileged in their ability to penetrate international boundaries by virtue of the knowledge and the use of Latin as a universal language; while the masses were united in the spiritual bond of a common religion, which helped to discipline their lives and make them reasonably contented with their lot. Ignorance, poverty and superstition were everywhere to be found, as well as a blind subservience to both Church and State. But the chance acquisition of new knowledge was bound, sooner or later, to disturb the old order of things.

The Seeds of Dissent.

At the beginning of the fourteenth century, a professor in the medical school of Padua, Pietro d'Abano (1250-1315), employed his scholarship in translating Greek medical and scientific works into Latin. Furthermore, his familiarity with the writings of Arabian physicians, particularly those of the philosopher and free-thinker Averroes (1126-1162), caused him to embark upon the dangerous venture of using Socratic methods of argument to reconcile medicine and religion. His literary activities soon came under the watchful eye of the Inquisition at Rome; but Nature stepped in before its machinery could be set in motion, and only his corpse could be committed to the flames.

Later in the century, John Wycliff (1324-1384), a leading Oxford scholar and an exemplary parish priest, began to translate the New Testament from the original Greek into the mother tongue. His work later set the standard for English prose. He then proceeded to give an English translation of the Old Testament from the Latin text of Saint Jerome. His thoughtful study of the Scriptures in the original context seemed to reveal certain basic truths which were strangely inconsistent with contemporary ecclesiastical views on the dogmas and practices of the Christian Church. He wished, therefore, to make these vital passages from the Gospel message available for the benefit of the English people.

Wycliff may have been indiscreet in his criticism of some aspects of ecclesiastical policy and conduct. He considered that these aspects deviated widely from the original tenets and requirements of the Christian faith as laid down by the early founders of his church. In due course, the Pope issued instructions for Wycliff to be imprisoned, but, owing to the peculiar temper and disposition of the English people, the order was quietly disregarded in official circles. Later on, Wycliff was summoned to the Papal Court at Rome to answer certain charges, but, once again, kindly Nature intervened. While celebrating Mass in his parish church at Lutterworth, he was stricken with a cerebral hæmorrhage, from which he died naturally on the next day. However, the matter was not allowed to rest here: thirty years later his writings were condemned as heretical and his bones were ordered to be taken from their resting-place and burned in the flames.

The Church and the State.

Dean Inge once remarked that the fruit of the tree of knowledge always drives man from some paradise or other. In the first half of the sixteenth century there gradually came about a reorientation of men's minds to new developments in the way of life. Both gunpowder and movable types were found to possess persuasive virtues of a subtle kind. The long-lost pagan literature and the Book of Holy Writ were translated, read, and found to be revealing. Besides all this, the mariner's compass had opened up an entirely new world previously undreamt of by any philosopher. The human spirit could no longer be held in bondage or oppressed by hoary tradition and ancient authority.

Now, the political and religious stability of Europe was profoundly disturbed by threats to end the much-abused despotisms of Church and State. Politics became impregnated with a strong religious flavour as a Protestant or Catholic regime would come alternatively into power; and that with as little concern to all but a few of the populace as a change of government might be viewed by us at the present day.

It was in the reign of Queen Elizabeth I that the new Calvinistic doctrines were introduced (1572) to sow discord and dissent among wavering adherents of the legally established Church of England. In Tudor times radical differences of opinion, in political or religious matters, were summarily adjusted by such drastic measures as beheading, burning, hanging, toturing or exile. These measures served to provide the world with many noble martyrs in the cause of freedom, and stored up a great deal of trouble for future generations.

An *Act of Supremacy* (1531) promulgated by Henry VIII had made it clear to the hierarchy in Rome that in England henceforth the Sovereign was to be head of the Church as well as head of the State. Queen Elizabeth I tried to abolish the papal powers in her country by her *Act of Uniformity* (1559) and by the *Conventicle Act* (1593). These Acts made it equally clear to all her people that only those subjects who conformed to the doctrines and practices of the Established Church would be entitled to the full privileges of citizenship.

As the first half of the seventeenth century came to a close, the dissenting and nonconformist elements had become so numerous and powerful that they were able to insist that the will of the people, as proclaimed through the voice of Parliament, must be paramount in framing and enforcing the laws of the land. So, for the time being, outworn acts of supremacy and uniformity had to go by the board. However, the bitter antagonisms of bygone days between the Church of England and the Church of Rome were stirred to the depths by the trends in world politics, and their protagonists were perpetually at daggers drawn.

[1] Read at a meeting of the Section of History of Medicine, Australasian Medical Congress (British Medical Association), Ninth Session, Sydney, August 20 to 27, 1955.

At the same time, minor differences in Protestant creed and form of worship had split the Nonconformists into Puritans, Baptists, Presbyterians, Congregationalists, and including an entirely novel and curious sect scornfully called Quakers (1650).

The Dæmon of Sectarianism.

During the second half of the century, religion and politics became intimately and inextricably interwoven into community life. Many thousands of respectable citizens spent their time in and out of gaol. Charles II and his parliamentary friends were fully occupied in paying off old scores and in dealing with recalcitrant Nonconformists. This they did in a series of statutes known as the *Corporation Act* (1661), the *Conventicle Act* (1664), the *Five Mile Act* (1665) and the famous *Test Act* in 1673. All these were superseded by *Toleration Acts* and *Declarations of Indulgence*, at a later stage, when the political wind began to blow in another direction. At the restoration of the monarchy, over two thousand dissenting preachers were turned adrift from their livings, without compensation, for not asserting their "unfeigned consent and assent" to the Thirty-nine Articles of Christian Faith as set forth unequivocally in the Book of Common Prayer (1563). More than twelve thousand members of the Society of Friends alone were thrown into prison, while others just as harmless were sentenced to transportation for work on plantations in the West Indies. In many high places there were confusion of thought, hypocrisy, endless intrigue and an almost sadistic mania for persecution to discover and punish unauthorized religious or political activities. Fanaticism, they say, consists in redoubling your efforts when you have already forgotten your aims. Our old friend, Sir Thomas Browne, wrote at that time: "The religion of one seems madness unto another."

The eighteenth century has been, rather optimistically perhaps, acclaimed as the Age of Reason, Toleration and Common Sense. At all events, these qualities frequently found expression in the Quaker way of life. The members of this peculiar sect harboured no resentment, but continued to work in harmony and brotherly love with the rest of the community. This was despite the fact that they were ridiculed, persecuted and deprived of many privileges and rights enjoyed by other citizens. In common with all dissenters, they were still debarred from attending the better schools and were excluded from the benefits of a higher education at the Universities of Oxford and Cambridge. They could not be legally married, except according to the rites of the Church of England, and were denied the right to hold any public or civic office.

When George II came to the throne in 1727, the Quakers still retained their unconventional mode of dress, an antique manner of speech, austere habits of living and a unique form of public worship; but they had abandoned all the former aggressiveness and were no longer seeking to gain proselytes and to promote popular revivalism. Now they were recognized as a quiet, sober, serious, inoffensive and highly respectable element in every British community. Indeed, their home training, religious upbringing and liberal education provided all the influences necessary for the making of industrious, useful and reliable citizens. Many promising avenues of public usefulness were still closed to the Quakers, but the more gifted ones sought careers in the business and professional world. As a class, they were noted for their humanitarian principles and for a readiness to extend practical Christian charity to those less fortunate than themselves; in fact, to all those in sickness, poverty or distress. Hence, the profession of medicine had a special appeal to many of their gifted sons.

Quaker Physicians.

The first, and perhaps the most famous of the Quaker physicians, was John Fothergill (1712-1780), who was born at Wensleydale in Yorkshire, just twelve months after the Tory leader, Lord Bolingbroke, had introduced the *Occasional Conformity Act*. This measure inflicted heavy fines on any person who had attained to State or Municipal office by taking the Sacrament in an Anglican church, and later had been discovered in the act of attending divine service in a Nonconformist place of worship.

Fothergill's early environment among the Society of Friends served to fit him admirably for a career dedicated to service in the new humanitarian age with its gradual leanings towards modern science. The main trend of his medical education was that customarily followed by Nonconformists for the next hundred years. At the age of sixteen years he became apprenticed to a surgeon in the provinces. Six years later he entered the medical school of Edinburgh University, where the Leyden tradition of teaching was being cultivated by Professor Alexander Monro. During his student days Fothergill distinguished himself as a scholar, and was one of the founders of the Royal Medical Society of Edinburgh, with William Cullen as one of his closest associates. After taking his degree in 1736, he went to London and spent two years in post-graduate work at Saint Thomas's Hospital under Sir Edward Wilmot.

In the year 1740, Fothergill made a short visit to several continental clinics. On his return to London he commenced practice at White Hart Court, Gracechurch Street, part of which building was occupied as an important meeting-house of the Friends. In these days he may have been too sensitive to apply for a public hospital position. He did not sit for his examination to qualify as a Licentiate of the Royal College of Physicians until 1744, when it was still considered an indulgence to grant even this diploma to a dissenter. In ordinary circumstances, he would be forbidden to practise as a consultant physician in the city and for seven miles around unless the College licence had been granted.

Within a few years, Fothergill became generally recognized as a leader in his profession. He wrote an authoritative book on scarlet fever in 1748, founded the Medical Society of Physicians in 1754 and was elected to the fellowship of the Royal Society of London for his pre-eminence as a botanist and horticulturalist. In the Royal Society *Transactions* of 1745 Fothergill recommended artificial respiration for the resuscitation of a person apparently drowned. In 1764 he founded the Society of Licentiate Physicians. The purpose of this society was to obtain redress of the wrongs suffered by those physicians who, on account of their nonconformity or of their holding a medical degree from a university other than that of Oxford or Cambridge, were ineligible to become fellows of the Royal College of Physicians of London. However, in spite of continued agitation, this privilege was not granted to licentiates until 1834. It is of interest to note that at the instance of his young friend, Joseph Banks, Fothergill with the able assistance of a fellow physician, Dr. Nathaniel Hulme, did all the spade work in providing Lieutenant James Cook, R.N., with the necessary knowledge and equipment for the great experiment in preventive medicine, which was carried out so successfully on Cook's first voyage of exploration to the South Seas (1770).

John Coakley Lettsom (1744-1813), a young Quaker from the West Indies, was much gifted with a ready wit, boundless energy, an enthusiasm for any good cause, an unerring sense for publicity, and the gracious manner of showing an interest in the welfare of his fellow creatures. From the day in 1761 that he decided to embark upon a medical career, it became his lifelong ambition to follow in the footsteps of the great Dr. Fothergill both as a man and as a physician, although in outlook and disposition their natures were as poles apart. Lettsom first served a long apprenticeship under a scholarly surgeon at Settle in Yorkshire. He then took Fothergill's advice in serving as a dresser in the wards of Saint Thomas's Hospital. He obtained his medical degree from the University of Leyden in 1769, then passed his examination for the diploma of the Royal College of Physicians of London and began practice at Gracechurch Street in 1770.

From his work among the poor in their hovels or places of incarceration, where he entered fearlessly at the risk of his life, he at once saw the need of an institution to serve this type of patient. So he founded the Aldersgate Dispensary with a staff of consultant physicians and surgeons

to work on a roster system in the out-patient department, domiciliary treatment being provided for those unable to attend. This kind of service set the pattern for similar institutions in London and elsewhere. In 1773, he was elected a Fellow of the Royal Society of London, perhaps for his botanical knowledge, his popular writings on tea and its use as a beverage, or for his estimable book "Naturalist's and Traveller's Companion", which was published in 1772. Above all, Lettsom is remembered for his founding in 1773 and his continued devotion to the Medical Society of London. The object of this society was to bring together all branches of the profession in order to promote new knowledge and mutual understanding. His professional, scientific and public activities were tremendous, while many of his good causes have prospered and stood the test of time. With Fothergill's prestige always behind him, Lettsom took an active part in founding in 1774 the Royal Humane Society, which first taught and practised methods for resuscitating the apparently drowned. He took up the work of his master in helping to prepare public opinion for the abolition of slavery and in giving full support to their mutual friend, John Howard, in his efforts to bring about prison reform. Finally, Lettsom was the originator in 1796 of a novel scheme to build a convalescent hospital or sanatorium at the seaside resort of Margate. The object of this institution was to hasten the recovery of tuberculous (scrofula) patients from the London hospitals and slums. Lettsom also used his powerful influence to gain recognition for Edward Jenner's discovery, by endeavouring to popularize vaccination against smallpox.

Two Lesser Lights.

It cannot be affirmed that all Quaker doctors were equally imbued with that "inward light" which gave Fothergill, Lettsom, Joseph Lister and Jonathan Hutchinson their idealistic conception of the duties and responsibilities rightly demanded of them as medical practitioners and public-spirited citizens of the world. Two contemporaries of Fothergill were each connected with a single event of some historical importance; although in each case the central figure fails to measure up to the high standard of professional efficiency customarily set by consultant physicians of the "connexion" as part of their normal and religious obligation to society.

Largely by virtue of his descent from a line of Quaker doctors, Thomas Dimsdale (1712-1800) managed to succeed as a surgeon at Hertford before acquiring, with the idea of becoming a physician, a medical degree from the University of Aberdeen. In 1767 he learnt the new technique of inoculation from its originator, Daniel Sutton, and twelve months later published his own book on the subject so that his reputation spread far and wide. On the recommendation of his friend, John Fothergill, he decided to risk his life or gain possible enduring fame by undertaking the long journey to Saint Petersburg in 1768 at the request of the Empress Catherine II. The object of this journey to Russia was to inoculate the Empress and her son, the Grand Duke. Fortunately for Dimsdale, the procedure of taking fresh serum from the pustule on the fourth day proved an unqualified success, and many others passed through the ordeal without a single fatality. With a sigh of relief, he gave vent to his feelings: "She has had the smallpox in the most desirable manner, which now, thank God, is over."

A somewhat eccentric and unprincipled member of the Society of Friends named Samuel Leeds managed to obtain a medical degree from the University of Edinburgh in 1766. Two years later he had himself appointed to the staff of the London Hospital as a physician. It was alleged subsequently that both these achievements were made possible by his creating the erroneous impression that he was a friend of Dr. Fothergill. However, there was a condition that the hospital appointment might be retained so long as Leeds took steps within a reasonable time to obtain the diploma of the Royal College of Physicians.

It was not long before Fothergill came to hear about the existence of Leeds, including several complaints of his ignorance and incompetence in the hospital practice, so he began to move swiftly in the matter. The governors of the hospital then demanded that Leeds take immediate steps to qualify for the diploma of the Royal College of Physicians or else resign from his position. After two attempts, Leeds was unable to pass the test. For the next two years, Fothergill suffered many harrowing experiences in defending himself before special courts set up by the Quakers, to hear the charges persistently followed up by the plaintiff, Leeds, for defamation of character and loss of professional status. At last, just as the Quaker tribunals had decided to give the decision against him, Fothergill was relieved to find himself faced with a hearing before the Court of King's Bench. There he won the appeal after the Lord Chief Justice, Mansfield, had completely exposed Leeds for an ignorant and incompetent medical practitioner. The outcome of this case was that, in future, all applicants for the appointment as a physician to the staff of any teaching hospital in Great Britain must be either a Fellow or a Licentiate of the Royal College of Physicians of London. Perhaps there may have been sufficient justification for the conservative attitude of the high and mighty at Warwick Lane in refusing to amend some of their archaic regulations.

The Rise of Rationalism.

Towards the end of the eighteenth century there was a greater tendency for medical men, who had been brought up to conform strictly with the Quaker code, to allow themselves some relaxation in minor points of discipline. Some went so far as to marry outside the "connexion", with the consequence of being disowned. Others transferred their allegiance to the Church of England which both opened up the way for many privileges essential to the progress of a consultant physician and gave them the liberty to enjoy more of the social pleasures of life.

After spending the first twenty years of his life within the Quaker fold, Thomas Young (1773-1829) thus drifted gradually away from the Society of Friends, from the time that he was persuaded by his uncle, Dr. Richard Brocklesby, to succeed him in his practice as an eminent London physician. It was unfortunate that medicine was then far from being an exact science, and that Young was probably the greatest intellectual genius that this world has ever seen, not excepting Aristotle, Avicenna and Albrecht von Haller. He was the eldest of 10 children born at Milverton in Somerset, and though encouraged in his studies by his Quaker parents, he never at any stage of early learning needed the instruction of a teacher. By the time he reached the age of nineteen years, he had attained professorial standard in Greek, Latin, some Oriental languages, mathematics and physics, but would have failed dismally as a teacher as he seemed incapable of descending to the intellectual level of his audience. When Young in 1792 commenced his medical studies at the Great Windmill Street School of Anatomy under John Hunter, Matthew Baillie and William Cruickshank, he was already a minor celebrity in the highest and more select academic circles of Great Britain. Next year, as a student at Saint Bartholomew's Hospital, he was given the eye of an ox to dissect, and the fresh specimens provided him with much food for thought. His paper, "Observations on Vision", read before the Royal Society of London in May of that year, deeply stirred the feelings of John Hunter and Everard Home, who claimed priority for the discovery of visual accommodation. For some years this formidable opposition shook Young's confidence in his theory, but the Royal Society, quite rightly as it turned out, vindicated his thesis by electing him a Fellow under the sponsorship of some of the greatest scientists of the day, shortly after his twenty-first birthday (June 13, 1794).

There is something strange, even pathetic, in Young's ambitious pursuit of fame as a distinguished London physician. He began the long climb by studying for the next two years at Edinburgh and Gottingen, during which period he slowly fell from grace as a conscientious Quaker, at least to all outward appearances. He had grown into a handsome man of fine physique with a pleasing manner, but severely discriminating in the exercise of his social charms. He valued the warm friendship of a few of his

male contemporaries and of many distinguished personages senior to him in age and position. He revelled in the society of attractive and intelligent women, learned to dance with ease and elegance, took lessons in playing the flute and clavicord, and learnt enough about voice production for him to entertain a select company with a good song to his own pianoforte accompaniment. After obtaining his medical degree from the University of Gottingen in 1797 with a thesis on the conservation of energy in the human body, a theme upon which Helmholtz elaborated fifty years later, he entered Emmanuel College, Cambridge, as a Fellow Commoner. He took this step in order to comply with the statutes of the Royal College of Physicians that a candidate for the diploma must spend two consecutive years at the same university before taking the degree. In Young's time the statutes required that six years must elapse between the admission of a student and his qualifying for the degree of Bachelor of Medicine, while another five years had to pass before the doctorate could be attained. Although Young started practice in London as a physician in 1799, presumably with the College Licentiate diploma, his Cambridge degree did not become effective until 1803, and it was another five years before he became a doctor of medicine and a Fellow of the Royal College of Physicians of London. Meanwhile he accepted the appointment as Professor of Natural Philosophy at the Royal Institution with Humphry Davy as a colleague. However, he resigned two years later because of an obsession that the public would come to associate his name with other departments of learning than that of medicine. After he was appointed physician to Saint George's Hospital in 1811, this peculiar foible gave rise to much misunderstanding and embarrassment in high literary and scientific circles throughout Europe, as he persisted for over eleven years in maintaining a rigid anonymity for all his articles in the "Encyclopædia Britannica" and in many important journals. Medical books and papers alone were allowed to be published under his name; so that his right to priority in the new theories of light and sound, his remarkable discoveries in physiological optics, and in the decipherment of the long-forgotten language of ancient Egypt were each frequently challenged both at home and abroad.

The rich legacy of learning bequeathed by Thomas Young, M.D., F.R.C.P., to literature, medicine, mathematics and the allied sciences was acknowledged by the British people shortly after his death, when an inscribed tablet surmounted by a profile medallion was placed in Westminster Abbey.

A Parting of the Ways.

In the twenties of last century the Nonconformist bodies in Britain, now considerably augmented by the Methodists and Unitarians, had become numerous and influential, and they were powerful enough to move for the establishment of a non-sectarian university within the city of London. As the initial result of their efforts, University College in Gower Street was opened in 1828 with a Faculty of Arts and Law, and a Faculty of Medicine, but theology was excluded from its curriculum. Shortly afterwards, King's College came into existence with a slight modification in its constitution to appease the religious sensibilities of orthodox churchmen. For a long time neither of these experimental and highly successful institutions received any support from the public purse.

The last of the Quaker physicians to adhere rigidly to the outward semblances and tenets of his sect was Thomas Hodgkin (1798-1866), who took his medical degree at Edinburgh in 1823. He became a Licentiate of the Royal College of Physicians two years later, and for the next decade worked quietly and unobtrusively as pathologist on the staff of Guy's Hospital. He visited the Continent, where he absorbed new concepts on the processes of disease and on physical diagnosis from the previous work of Bichat and Laennec. On his return he demonstrated the use of the stethoscope in the wards of his hospital. In his pathological studies at Guy's he anticipated Corrigan in a description of aortic insufficiency in 1827, published the first account of a disease characterized by a chronic enlargement of the lymphatic glands and spleen, and arrived at a clear understanding of acute appendicitis and its complications. He suffered a severe disappointment in 1837, when he failed in his candidature for appointment to the vacant position of assistant physician on the Guy's Hospital staff; but the post was given to a man holding a Cambridge degree and the Fellowship of the Royal College of Physicians. Hodgkin thereupon tendered his resignation as pathologist, entered private practice for a time in London, and then devoted the remainder of his life to the pursuit of charitable objects in the distinguished company of Sir Moses Montefiore, the noted Jewish philanthropist.

With the passing of the *Medical Act* of 1858, all undergraduates of the teaching schools received the qualifying diploma of Licentiate of the Royal College of Physicians, while graduates of a recognized university were eligible to receive the membership diploma after passing a higher examination in medicine. Another effect of this legislation was that for the first time in centuries the Church was completely dissociated from all statutory enactments relating to the profession of medicine. Twelve years later in 1870, all Nonconformists were given the freedom to compete on equal terms with the rest of the community.

> The test of every religious, political and educational system is the man which it forms.—AMIEL.

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References.

ABRAHAM, J. J. (1933), "Lettsom: His Life, Times, Friends and Descendants", London.

GREEN, J. R. (1915), "A Short History of the English People", London.

FOX, R. H. (1919), "Dr. Fothergill and his Friends", London.

HOOKER, J. D. (1896), "Journal of the Right Hon. Sir Joseph Banks".

OLDHAM, F. (1933), "Thomas Young, F.R.S.: Philosopher and Physician", London.

PEACOCK, G. (1855), "Thomas Young, M.D., F.R.S.", London.

PEACOCK, G., and LEITCH, J. (1855), "The Miscellaneous Works of Thomas Young", London, 3 vols.

PETTIGREW, T. J. (1838), "Medical Portrait Gallery", Vols. 3 and 4.

TREVELYAN, G. T. (1945), "History of England", 3rd edition, London.

TREVELYAN, G. T. (1946), "English Social History", 2nd edition, London.

WILKS, S., and BETTANY, G. T. (1892), "A Biographical History of Guy's Hospital", London, 380-386.

WOOD, A. (1954), "Thomas Young: Natural Philosopher, 1773-1829", University Press, Cambridge.

THE MAJOR PSYCHIATRIC PROBLEMS OF ADOLESCENCE.[1]

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MATERNAL DEPRIVATION in early childhood and the after-effects of the broken home are now recognized as important factors in psychiatric disability. Many of the men and women left by their fathers in the 1914-1918 war suffered the effects of family disruption in early life (Stott, 1950). Their own children were born just before or during the second World War, and these children in their turn suffered similar deprivations, which in the European countries were aggravated by bombing and mass evacuation. Now this second generation is growing up, and we are seeing the effects of a double deprivation, the deprived children of deprived parents.

[1] Read at a meeting of the Section of Neurology and Psychiatry, Australasian Medical Congress (British Medical Association), Sydney, August 20 to 27, 1955.

It would be of interest to see if the parents of the present-day disturbed or delinquent adolescents had similar episodes at a corresponding period of their own youth. The time factor is of importance, for the incidence of crime, Sir Sydney Smith (1952) pointed out, rose alarmingly in the sixteen-year-olds and under, in the twelve years after the 1918 war; and it remained at so high a level that in 1950 no less than 2% of all fourteen-year-old boys in England appeared in court.

Is the same pattern to be followed again with the same time relationship to the second war? Is there likely to be a further increase in delinquency in the next few years, with sexual promiscuity in the females and larceny and housebreaking in the males? Smith indicates that the child is born a potential criminal, aggressive and acquisitive, but in a normal family learns by example to curb his antisocial tendencies and to absorb the culture of the society to which he belongs.

But what of the adolescent coming from a broken home, brought up without a father, or the one who is rejected or subdued all his life? It is easy for him to be faced with emotional situations he cannot handle and for delinquency to result, as he has neither the experience to deal with his problems nor the security necessary to make decisions. He must flee from his conflicts and seek relief from his anxieties, as did so many of the boys in the series of delinquents examined by Stott. Fortunately, even with so poor a start comparatively few of the juvenile delinquents carry their deficiencies to adult life, and Bovet (1951) estimated their continuance at no more than 10% to 20%.

Hutchinson's (1954) social study of the aged in Victoria shows the greater facilities available for the care of the aged in the consanguine family, which is based on blood relationships. In such a family there is also a close supervision of the children. On the other hand, the modern conjugal family is made up of the man, the wife and their offspring, and here the emancipation and independence of the children are all important. The tendency is, therefore, established to free the young adolescents at so early an age that they are unable to meet their problems with the necessary foresight. So often these adolescents must find their recreations at the cinema and fun-parlours, their reading in the comics and paper-backed novels, and their work in highly paid dead-end jobs.

But, as Bowlby (1949) and Lewis (1954) and others have shown, only a proportion of those who have been deprived and neglected break down. Because a high percentage of the adolescent delinquents, neurotics and schizophrenics and others who have behaviour problems come from broken homes, this does not mean that a broken home will invariably produce these disabilities; in fact, perhaps not more than a third of such children show these disorders. There is, therefore, room for long-term experiments, beginning with the investigation of the disrupted family and then following the offspring to see if they become emotionally disturbed as they grow up. Nevertheless, according to Bowlby, the child's early social environment is relative in a highly significant degree to an understanding of later social behaviour, and the children from broken homes are, therefore, potential social casualties.

Observations on maternal deprivation, rejection and hostility by Bowlby (1951), Stott and others must be regarded as of the greatest importance to mental hygiene. But I wonder if we may not have under-estimated the role of the father in preventing psychiatric disabilities, first in regard to post-war adolescent problems and perhaps as well in juvenile psychoses. It may even be that a tendency will be found for one type of disturbance to occur in the children with maternal deprivation, and another when there is paternal absence or neglect.

Mental Deficiency.

Amongst the many advances in child psychiatry in the past twenty years those in the maternal deficiency field are of much importance. For instance, "simple defectives" used to be said to constitute 75% of the total, but amongst these have been found cases due to German measles in the mother (Gregg, 1941), erythroblastosis (Evans and Polani, 1950), lead poisoning, phenyl pyruvic oligophrenia (Folling, 1934) and other causative factors; whilst the traumatic deficiencies have been clarified by the better knowledge of cerebral palsy and the concept of neurophrenia in other injuries (Doll, 1951). Now juvenile schizophrenia must also be considered. For a number of years Tredgold (1945) emphasized the relationship between the simple schizophrenic and the mental defective, tending to regard the psychotic as a mental defective. Recent work has suggested the same in reverse, which is that many institutional mental defectives are in reality schizophrenics.

O'Gorman (1954) has collected observations in this subject made during the past fifty years, though juvenile schizophrenia has only recently been recognized as a factor of appreciable magnitude in producing a "de-facto" mental defect before the age of eighteen years. There are many cases of apparent defect in which children are brought for advice with a history of the condition having come on after some years of normal development. Sometimes their history either has been doubted on the grounds of the mother's emotional involvement or has been put down to a cerebral infection of doubtful origin; in fact it seems that these may often be suffering from schizophrenia.

The relationship between schizophrenia and mental deficiency has not, in the past, been clear. At one time it was taught that the mental defective and particularly the unstable feeble-minded adolescent was susceptible to schizophrenic-like episodes of good prognosis, and these were regarded as due to the patient's instability. On the other hand, because of their subnormal intelligence it was thought that the mental defectives were likely to develop a simple schizophrenic illness with a poor prognosis. Perhaps it was a recrudescence of an established process on the one hand and a chronic schizophrenic illness which had increased in intensity in adolescence on the other.

Adolescent Schizophrenia.

There is also evidence to suggest that many of the schizophrenic psychoses which occur in adolescents of normal intelligence have had their origin in childhood. Neubauer and Steinert (1952), for instance, believe that in adolescent schizophrenia the disease may have been present since childhood or earlier, and that a careful history may show that previous anxiety states have flared up from time to time and been misdiagnosed as psychoneurotic disorders. Spontaneous recovery from these attacks usually takes place, but there is a possibility of increasing the anxiety if they are treated as being due to a neurosis. However, because a child has been very shy and withdrawn the chances of schizophrenia developing in adolescence or after are not as great as might be supposed. This was shown by Morris, Soroker and Burruss (1954) in their figures from the follow-up of patients who had first attended a child guidance clinic years before, only 4% becoming psychotic in later life.

Adolescent schizophrenia has been known for years, even to Craig's dictum of "wrecked on the rocks of Puberty", but pre-adolescent schizophrenia has been slow to be recognized. Teaching on adolescent schizophrenia has therefore tended to follow the lines of the adult disorders, instead of being related to childhood development.

It has now been shown, particularly by Bender (1953) that childhood schizophrenia frequently occurs, and in the past twenty years she has established criteria for the diagnosis of schizophrenia before the age of eleven years. She has followed many patients through into adult life, when two-thirds are still found to be suffering from the condition. Bender suggests that there is a strong hereditary factor in childhood schizophrenia, and that the subjects show primitive homœostatic control, though the pattern of the illness tends to be governed by the parent-child relationship.

Other writers, such as Melanie Klein, Despert (1938), Creak (1951) and Clardy (1951) have mentioned the frequency of its occurrence, the confusion in diagnosis,

the dubious prognosis, its development and the part that the parent-child relationship has played in its pattern. Oltman, McGarry and Friedman (1952) found no increased incidence of parental deprivation or broken homes in the histories of schizophrenic children, though the parents of these children were frequently found to have been mentally ill. However, Wahl (1954) found parental rejection or over-protection and/or parental loss in 88% of cases.

Wilson (1951) gives a most useful account of the onset and development of schizophrenia in adolescents, based on the early deviations from their normal psychological make-up, but complicated by all the vicissitudes of their reactions in bridging the gap from childhood to adult behaviour. He stresses the fact that many of the normal reactions in adolescence are not unlike the symptoms of the schizophrenic, and he shows the necessity for balance, security and stability in the home to aid the girl or boy in making adjustments.

It appears that much may be gleaned from a study of the schizophrenic-like reactions seen in patients in general practice. Psychotic episodes of short duration are probably not uncommon and are often overcome by sedation; but psychiatrists, particularly in mental hospital practice, very rarely have the opportunity to see these illnesses, of which the significance may not be fully known.

Burns (1952) described two special types of maladjustments in intelligent schoolboys. The one occurs between the ages of fourteen and sixteen years, with withdrawal and feelings of unreality; this is comparable to a more florid reaction in girls of the same age. The other occurs in the hypersensitive, mother-fixated types of boys, who have difficulties at school and corresponding phobias. The prognosis in both groups is, with intelligent management, better than might at first be expected.

Although the prognosis may not be good in cases of established schizophrenia in adolescence, it is not as gloomy as may be supposed; and there is some evidence that, if glandular deficiencies are overcome with androgen-œstrogen therapy, a number of subjects may do better than with the usual physical methods of treatment.

Electroencephalographic Findings.

The electroencephalographic findings in adolescence have not yet been clarified. It has been found that about a third of all schizophrenics show non-specific abnormal records and that their frequency is greater in the relatives of the schizophrenics than in the normal population. Moreover, siblings of schizophrenic patients with abnormal records show a much higher incidence of abnormality than do the siblings of parents with normal records (Chamberlain and Russell, 1952). It has already been shown that there is a greater incidence of abnormal electroencephalograms in young schizophrenics than in adult subjects; therefore it has been suggested there are organic changes in early schizophrenia.

Schizophrenic-like symptoms are found in some cases of epilepsy in childhood, and temporal lobe epilepsy has sometimes been difficult to distinguish from schizophrenia. Perhaps this is because automatism is particularly common with temporal foci (Liddell, 1953), and perseveration of thoughts and emotions is characteristic of their symptomatology (Alstrom, 1950). Robertiello (1953) investigated psychomotor epilepsy in children and found impulsive erratic periodic behaviour disorders comparable in their violence and bizarrity to a schizophrenic symptomatology. On the other hand, Pond (1952), writing of adults, could find no connexion between epileptic and schizophrenic symptoms, and found that frank psychotic episodes were rare. When these disturbances occur in adolescent females, they must be carefully charted; for not infrequently the epileptic, migrainous or behaviour disturbances are found to be related to a definite stage of the menstrual cycle, and they will often respond to progesterone therapy.

Additionally, disorders of behaviour in which abnormal electroencephalograms occur are in some cases associated with enuresis, moodiness, irritability and masturbation (Hill, 1947). The patients often improve on treatment with amphetamine. Other patients with abnormal rhythms show signs of emotional and social immaturity. Speculation on the possible relationship of immaturity, psychotic symptom formation, behaviour disorders and psychomotor epilepsy to abnormal electroencephalographic findings would appear to be of particular interest in the developing adolescent.

Manic-Depressive Disorders.

Of recent years, Campbell (1952, 1953) has called attention to the manic-depressive disorders of childhood, and these were most usefully reviewed in a volume of "The Nervous Child" devoted to this purpose. It has previously been supposed that psychotic depression was rare and mania was almost unknown (McHarg, 1954), although Sadler in 1948 mentioned a number of cases. Campbell has shown that many cases of melancholia in later life can trace their depression back to childhood in some cases and to adolescence more frequently. It is found that they have conformed to a type which is serious, likeable, intelligent and cooperative. They have been given responsibility, but they have worried through their conscientiousness and desire to succeed. The cyclothymic types are commoner, but their sensitivity and the ease with which they show ideas of reference when disturbed may, in early life, have tended to confuse their diagnosis with that of schizophrenia or psychoneurosis.

Adolescents show many depressive features which are recognizable in any series of their paintings; such features are also to be found by personality testing, as, for instance, with the thematic apperception test or Symonds (1949) picture tests. Guilt reactions, self-blame and remorse are often found in association with, for example, masturbation.

Suicide is not common in adolescence, but it is sometimes attempted by young depressives, and the attempts are often closely related to imagined loss of parental love (Despert, 1952). Suicide may seem to be the only solution to what appear otherwise to be insoluble emotional problems. Suicide in schizophrenia is better known because the bizarre methods chosen often lead to publicity.

Incidence and Family Background.

Levy and Southcombe (1951) found 291 cases, 2% out of 14,858 mental hospital admissions, to be in patients aged eighteen years or less, of whom 103 or 35% were schizophrenics. From the records of a recent series of 4556 consecutive admissions to Royal Park Hospital, 2·1% of the cases were in patients aged eighteen years and less (of whom a quarter were aged twelve to fifteen years and 54% were males). If we exclude the 13 grosser mental defectives, 45% of the remainder were schizophrenics, but only 7% suffered from an affective psychosis. It is interesting that suicide had been attempted by 15% of the adolescents from Royal Park.

Thirty-nine cases were reviewed in which full social histories were available; in four of these the mother was psychotic, in two the father, and in a further two cases both parents. In seven the father had deserted the mother, died or been separated from the mother in the patient's early childhood, and in a further six the father was a drunkard. In another six cases there was considerable family inadequacy or disruption. In all, these cases constituted a formidable proportion of the series, being 27 of the 39 cases chosen. However, this method of selection probably loads the series a little towards those patients having particular family difficulties.

Conclusion.

I hope that this short review may have shown that there are many psychiatric problems of adolescence which are unsolved. The time would seem opportune for inquiries to be made into the antecedents of adolescent disturbances and the frequency of their occurrence. The differential diagnosis of the psychoses in childhood and adolescence is often obscure, and there is confusion between childhood schizophrenia and a number of other conditions. The symptomatology of the adolescent disorders needs descrip-

tion—particularly because they are coloured by a peculiar mixture of independence and dependence, rebellion and submissiveness, over-confidence and insecurity. For instance, how far can the suggestibility, the quick suspicions, the emotional extremes, the anguish, the self-blame, the secret fears, the day dreams and the unpredictability be recognized as the normal background which complicates abnormal behaviour?

Much of interest has come from the electroencephalographic findings in some cases of disordered behaviour. But they form a series of isolated observations rather than a related pattern.

The affective disorders often have a much longer history than is usually supposed, and the recognition of cyclothymic tendencies in adolescence would be valuable in preventing later illness.

There seems to be little doubt that the major psychiatric disorders are of greater frequency and importance in childhood and adolescence than was previously supposed. Their description has, however, been static through failing to allow for the changes associated with maturation, but there is already evidence that research in this field is yielding many discoveries of significance.

References.

ALSTRÖM, C. H. (1950), "A Study of Epilepsy", Munksgaard, Copenhagen.

BENDER, L. (1947), "Childhood Schizophrenia: Clinical Study of 100 Schizophrenic Children", *Am. J. Orthopsychiat.*, 17: 40.

BENDER, L. (1953), "Childhood Schizophrenia", *Psychiat. Quart.*, 27: 663.

BOVET, L. (1951), "Psychiatric Aspects of Juvenile Delinquency", World Health Organization, Geneva.

BOWLBY, J. (1949), "Why Delinquency?", National Association for Mental Health, London.

BOWLBY, J. (1951), "Maternal Care and Mental Health", World Health Organization, Geneva.

BOWLBY, J. (1953), "Some Pathological Processes Set in Train by Early Mother-Child Separation", *J. Ment. Sc.*, 99: 265.

BURNS, C. (1952), "Pre-Schizophrenic Symptoms in Pre-Adolescents' Withdrawal and Sensitivity", *Nerv. Child*, 10: 120.

CAMPBELL, J. D. (1952), "Manic-Depressive Psychosis in Children", *J. Nerv. Ment. Dis.*, 116: 424.

CAMPBELL, J. D. (1953), "Manic-Depressive Disease", Lippincott, Philadelphia.

CHAMBERLAIN, G. H. A., and RUSSELL, J. G. (1952), "The E.E.G.'s of the Relatives of Schizophrenics", *J. Ment. Sc.*, 98: 654.

CLARDY, E. R. (1951), "Study of Development and Course of Schizophrenia in Children", *Psychiat. Quart.*, 25: 81.

CREAK, E. M. (1951), "Psychoses in Childhood", *J. Ment. Sc.*, 97: 545.

DESPERT, J. L. (1938), "Schizophrenia in Children", *Psychiat. Quart.*, 12: 366.

DESPERT, J. L. (1952), "Suicide and Depression in Children", *Nerv. Child*, 9: 378.

DOLL, E. A. (1951), "Neurophrenia", *Am. J. Psychiat.*, 108: 50.

EVANS, P. R., and POLANI, P. E. (1950), "Neurological Sequelæ of Rh Sensitization", *Quart. J. Med.*, 19: 129.

FÖLLING, A. (1934), "Phenylketonuria", *Nord. Med.*, 8: 1054.

GLASSER, F. B., *et alii* (1951), "Relation of Rh to Mental Deficiency", *Psychiat. Quart.*, 25: 282.

GREGG, N. McA. (1941), "Congenital Cataract following German Measles in the Mother", *Tr. Ophth. Soc. Australia*, 3: 35.

HILL, D. (1947), "Amphetamine in Psychopathic States", *Brit. J. Addict.*, 44: 50.

HUTCHINSON, B. (1954), "Old People in a Modern Australian Community", University Press, Melbourne.

LEVY, S., and SOUTHCOMBE, R. H. (1951), "Value of Convulsive Therapy in Juvenile Schizophrenia", *Arch. Neurol. & Psychiat.*, 65: 54.

LEWIS, H. (1954), "Deprived Children", Oxford University Press, London.

LIDDELL, D. W. (1953), "Observation on Epileptic Automatism in a Mental Hospital Population", *J. Ment. Sc.*, 99: 732.

MCHARG, J. F. (1954), "Mania in Childhood", *Arch. Neurol. & Psychiat.*, 72: 531.

MORRIS, D. P., SOROKER, E., and BURRUSS, G. (1954), "Follow-Up Studies of Shy Withdrawn Children: 1. Evaluation of Later Adjustment", *Am. J. Orthopsychiat.*, 24: 743.

NEUBAUER, P. B., and STEINERT, J. (1952), "Schizophrenia in Adolescence", *Nerv. Child.*, 10: 129.

O'GORMAN, G. (1954), "Psychosis as a Cause of Mental Defect", *J. Ment. Sc.*, 100: 934.

OLTMAN, J. E., MCGARRY, J. J., and FRIEDMAN, S. (1952), "Parental Deprivation and 'Broken Home' in Dementia Præcox and Other Mental Disorders", *Am. J. Psychiat.*, 108: 685.

POND, D. A. (1952), "Psychiatric Aspects of Epilepsy in Children", *J. Ment. Sc.*, 98: 404.

ROBERTIELLO, R. C. (1953), "Psychomotor Epilepsy in Children", *Dis. Nerv. System*, 14: 337.

SMITH, S. (1952), quoted in "Our Crime Sheet" (leading article), *Lancet*, 1: 1149.

STOTT, D. H. (1950), "Delinquency and Human Nature", Carnegie Trust, Dunfermline.

SYMONDS, P. M. (1949), "Adolescent Fantasy", Columbia University Press, New York.

TREDGOLD, A. F. (1945), "Manual of Psychological Medicine", Baillière, London.

WAHL, C. W. (1954), "Antecedent Factor in the Family History of 392 Schizophrenics", *Am. J. Psychiat.*, 110: 668.

WILSON, H. (1951), "The Early Diagnosis of Schizophrenia", *Brit. M. J.*, 1: 1502.

SARCOID-LIKE LESIONS IN LYMPH NODES DRAINING CARCINOMA.[1]

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THE disease entity described by Besnier (1889), Boeck (1899 and later) and Schaumann (1914 and later), and now known by various names, such as Boeck's disease, Boeck's sarcoidosis or simply sarcoidosis, is difficult to define.

At a conference on sarcoidosis in Washington (1948) it was decided to use the following rather broad descriptive "definition":

> Sarcoidosis is a disease of unknown etiology, characterised pathologically by epithelioid tubercles, with inconspicuous or no necrosis, occurring in any organ or tissue, and by the frequent presence of refractile or apparently calcified bodies in the giant cells of the tubercles. The lesion may be replaced by fibrosis, hyalinisation or both.
>
> Clinically the lesions may be widely disseminated. The tissues most frequently involved are the lymph nodes, lungs, skin, eyes and bones. The clinical course is usually chronic with minimal or no symptoms. However, there may be acute phases, characterised by malaise and fever. There may be symptoms referable to the tissues and organs involved. The tuberculin test is frequently negative. Plasma globulin is often increased. The outcome may be clinical recovery without gross or radiologically visible residuals, or it may be impairment of the function of the organs involved, or a continuous chronic course of the disease.

None of these described features is pathognomonic for Boeck's sarcoidosis, and a wide variety of agents can occasionally provoke a similar reaction (Lichtman, McDonald, Dixon and Mann, 1946).

The fact that in the foregoing description the lesions are called "tubercles" indicates that identical lesions can sometimes be produced by tubercle bacilli. In tuberculoid leprosy the lesions may be indistinguishable from sarcoidosis (Lever). In toxoplasmosis these lesions occur occasionally, and *lymphogranuloma inguinale* and cat-scratch disease may provoke them. Chemical substances like silica and particularly beryllium can induce identical changes. Typical lesions lead to the name of "giant-cell thyroiditis" and are found in cases of Riedel struma and *struma lymphomatosum*. Many more examples could be cited.

A diagram (Figure I) taken from a monograph by Olav Refvem demonstrates the main possibilities.

In many of these conditions, because of other histological changes, it is possible to make a correct diagnosis. The point which must be emphasized is that sarcoid-like tubercles can and do form in a variety of diseases.

[1] Read at a meeting of the Section of Pathology, Bacteriology, Biochemistry and Forensic Medicine, Australasian Medical Congress (British Medical Association), Ninth Session, Sydney, August 20 to 27, 1955.

Although these facts ought to be known by pathologists, it is still customary to diagnose "sarcoidosis" in lymph node biopsies when the described lesions are present in the absence of demonstrable acid-fast bacilli and in the absence of other obvious causes.

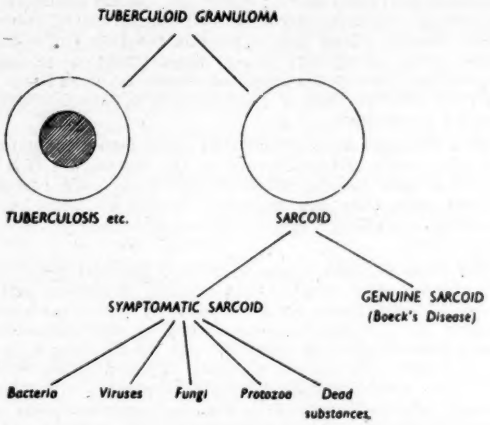

FIGURE I.

Illustrating schematically the main groups under the heading of tuberculoid granuloma. Circle with black centre, epithelioid cell granuloma with central necrosis. Open circle, epithelioid cell granuloma without central necrosis. (From Refvem, 1954.)

My interest in this question was evoked when several years ago I observed sarcoid-like lesions in three lymph nodes (Figure II) in the lesser omentum of a man, aged sixty-eight years, who died from obstructive jaundice as a result of a primary carcinoma of the main hepatic duct. No similar lesions were found elsewhere in the body; neither was there any indication of tuberculosis.

In 1950 Gherardi made a very similar observation on a woman, aged sixty-one years, who also died of obstructive jaundice as a result of carcinoma of the bile ducts. In this case the neighbouring lymph nodes were again the only foci in the body with sarcoid-like lesions. Gherardi considered this to be the first case in which there was a combination both of carcinoma and of Boeck's sarcoidosis.

In the same year Nadel and Ackerman described five cases of malignant disease in which sarcoid-like structures were found in the draining lymph nodes, and in 1951 Symmers described another five. Nickerson, in an article on sarcoid, described lesions in a retro-aortic lymph node in a case of carcinoma of the stomach, and it is probable that this falls into the same category. I have observed several more such lesions in lymph nodes draining breast cancers (Figures III, IV and V), and other pathologists have since supplied me with more material. In one of these cases further extensive changes were found in a couple of lymph nodes draining a primary carcinoma of the bile ducts (Figure VI).

The details are set out in Table I.

Although the site of the primary tumour varies considerably, it may have some significance that in this list there are seven cases of breast cancer, four of cancer of the bile passages and three of cancer of the bronchi, of which two were malignant "adenomata".

Actual sarcoid-like structures inside tumours have also been observed and are well known to occur in some cases of *seminoma testis* and ovarian disgerminoma (Schiller, 1934). The lesions have been described in a retroperitoneal neurocytoma (Hartz and van der Sar, 1945), and are such a constant feature in some cases of thymus tumour that this type of tumour is even called "granulomatous carcinoma of the thymus".

It is hardly conceivable that in all these cases the coexistence of malignant disease and sarcoid-like lesions is accidental. Far more likely is it that some product of the tumour itself is responsible.

This product may be due to the actual metabolism of the tumour, or it may be a breakdown product produced by degeneration or necrosis. With the possible exception of the above-mentioned cases, in which the lesions are found inside the actual tumour, it seems more likely that a breakdown product is involved as such a variety of causative tumours is found. This seems the more likely as in a considerable number of cases the primary tumour had been irradiated beforehand. However, an actual metabolic product cannot be excluded, as the majority of tumours were of a glandular origin (see list).

TABLE I.

Case No.	Site of Primary Tumour.	Diagnosis.	Site of Involved Lymph Gland.	Author.
1	Stomach.	(? Adenocarcinoma.)	Retro-aortic.	Nickerson.
2	Cystic and main hepatic ducts.	Adenocarcinoma.	Lesser omentum.	Gherardi.
3	Oral cavity.	Epidermoid carcinoma.	Anterior cervical.	Nadel and Ackerman.
4	Skin, left frontal region.	? Melanocarcinoma.	Left side neck.	
5	Left breast.	Scirrhous carcinoma.	Axilla.	
6	Ampulla of Vater.	Adenocarcinoma.	Minor omentum.	
7	Right main bronchus.	Malignant "adenoma".	Hilus of lung.	
8	Right breast.	Scirrhous carcinoma.	Axilla.	Symmers.
9	Right breast.	Scirrhous carcinoma.	Axilla.	
10	Thyroid gland.	Solid carcinoma in *struma lymphomatosum*.	Neck.	
11	Bronchus, lower lobe of right lung.	Anaplastic carcinoma.	Below azygos vein.	
12	Bronchus, upper lobe of left lung.	"Adenoma" cylindroma.	Alongside interlobar septa.	
13	Cystic and main hepatic ducts.	Adenocarcinoma.	Lesser omentum.	ten Seldam.
14	Cystic and main hepatic ducts.	Adenocarcinoma.	Lesser omentum.	
15	Left breast.	Scirrhous carcinoma.	Axilla.	
16	Right breast.	Scirrhous carcinoma.	Axilla.	
17	Right breast.	Scirrhous carcinoma.	Axilla.	
18	Left breast.	*Carcinoma simplex.*	Axilla.	
19	Uterus.	Adenocarcinoma.	Para-aortic.	

If we accept these cases of isolated sarcoidosis in lymph nodes as being of a reactive nature—in other words, sarcoid-like but not really sarcoid—the problem assumes some practical importance.

Nowadays it has become a common procedure to make scalenic lymph node biopsies to arrive at the correct diagnosis of intrathoracic lesions. The frequency with which sarcoidosis is diagnosed in these lymph nodes is clearly seen from the figures in Table II (kindly provided by Dr. Cotter Harvey).

It is self-evident that in a number of these cases real sarcoidosis as a systemic disease is the underlying cause; but it is my contention that in an appreciable number this may not be so, and that intrathoracic malignant disease may have provided the stimulus to a sarcoid-like reaction. To be aware of this possibility is of great importance to both clinician and pathologist.

The actual proof that sarcoid-like lesions can be provoked by tumour products must lie in experiment, and for this reason it was decided to investigate the possibility.

The Present Investigation.

As in a considerable number of reported cases the lesions were found in the axillary nodes of a primary breast cancer, and as this form of tumour is readily accessible it was decided to make most of the investigations with extracts of breast cancers, according to the following method.

Method.

The tumours were received in a fresh state shortly after surgical removal. They were surgically cleaned as nearly as possible of all macroscopic fat, then weighed and cut up into small pieces. These were rubbed with a measured amount of a physiological saline solution. The amount of saline solution used depended on the weight of the tumour, and varied from two to five millilitres per gramme of wet weight of tumour. The fluid was then filtered through several layers of gauze and had a turbid, sometimes rather milky appearance. Occasionally some fat droplets were seen floating on top of the fluid before filtration. These were carefully removed with a Pasteur pipette. All manipulations were made under sterile conditions. When the material was kept over a longer period at room temperature a fine sediment would form Particulate matter of visible size was not present.

TABLE II.
Scalenic Lymph Node Biopsies.

Diagnosis.	Royal Prince Alfred Hospital.	Royal North Shore Hospital.
Sarcoidosis	4	4
Carcinoma	4	5
Tuberculosis	—	1
Hodgkin's disease	—	1
Lymphoblastoma	—	1
Negative findings	8	33
Total	16	45

With the extract of each tumour six adult rats, mainly males, were injected intrasplenically with 0·5 millilitre and in some cases one millilitre. The rats were killed on successive days, the first one after four days in the first experiments, and after five days in later experiments.

Results.

In all rats reaction in the spleen was surprisingly small. An area of slight fibrosis with numerous hæmosiderin-laden macrophages around the site of injection was a constant finding, but no lesions resembling sarcoidosis were found.

Naked-eye examination of the liver usually revealed very little abnormality, though occasionally pale areas giving a mottled appearance, particularly of the left lobe, were observed.

In a few instances the histological examination of the liver revealed small lesions in or near the portal tracts. These lesions consisted of an epithelioid reaction with or without giant cells of the Langhans type, and contained surprisingly little lymphocytic reaction (Figures VII and VIII). Occasionally smaller lesions were found consisting of accumulations of lymphocytes (Figure IX). In some lesions a pointed, slit-like space was present in the centre (Figure X), but this was not a constant finding.

The nature of this space was not clear. It could hardly be of protein nature, as it took no stain whatsoever. The possibility has to be considered of cholesterol, perhaps formed as a result of the traumatic hæmorrhage in the spleen due to the injection, or it could be some particulate matter from the extract. However, the lesions were completely absent in the control series injected with normal saline. As the lesions were found only in some of a number of serial sections it was not possible to make frozen sections.

The possibility exists that the described lesions are nothing else than foreign body reactions around tiny particles which escaped the filtering property of the spleen. Even so, the results seemed to warrant further investigation.

Sabin *et alii* and Anderson demonstrated that the phospholipid fraction of tubercle bacilli induces the epithelioid reaction and giant-cell formation of tubercles, while Stanley found that a similar reaction could be provoked with phospholipids extracted from a pathogenic *Aspergillus fumigatus* isolated from a penguin. Olaf Refvem believes that a phospholipid is also involved in Boeck's sarcoidosis.

In a research on the nature of the asteroid inclusions of the giant cells, Hirsch comes to the conclusion that they are of a lipid nature, while Jaques gives good reasons to believe that they are at least related to phospholipids, possibly resulting from some disturbance in lipid metabolism.

For these reasons it was decided to proceed with further experiments with phospholipid extracts of tumour material. In the Department of Pathology of the University of Sydney Mr. J. B. Raby has been working with such extracts, using various sites of injection, but up till now with very little result. In one of his rats, injected subcutaneously with an alcoholic extract, small abscesses were formed around which fibrosis with giant-cell reaction took place, but nothing resembling sarcoidosis was present. Severe fibroblastic reactions were repeatedly observed in other animals, but the draining lymph nodes showed no specific reaction.

Discussion.

Even though the experimental evidence up to date is rather disappointing, a relationship between sarcoid-like lesions in certain lymph nodes and a carcinoma in the region which they drain cannot lightly be discarded.

The observations mentioned at the beginning of this paper could easily be multiplied many times if all pathologists would go through their records with this object in mind. Even so, the combination is far from constant and occurs only in a minority of cases of malignant disease. This indicates that only certain tumours will produce the necessary substance, or perhaps that blockage of the lymphatics by tumour tissue prevents the substance from reaching the draining lymph nodes. Olaf Refvem believes that sensitization may play a part in the cause of the lesion of sarcoidosis. Whatever the cause, further research seems warranted, and a study of the phospholipid content of certain tumours will be our next step.

A possible correlation between sarcoid-like lesions in scalenic lymph nodes and intrathoracic disease also needs further investigation.

Summary.

1. The literature on the presence of sarcoid-like lesions in lymph nodes draining an area affected with carcinoma is briefly reviewed and a number of observations are added.

2. As a wide variety of conditions can provoke sarcoid-like lesions it is stressed that probably products of necrosis or metabolism of certain tumours cause these lesions in the regional lymph nodes.

3. It is pointed out that this relationship has assumed a practical importance since scalenic lymph-node biopsies have become fashionable, and in a fair number of these lymph nodes the lesions are found.

4. The probability that these lesions are produced by a phospholipid is discussed, and the results of preliminary experiments to induce the lesions in animals are reported.

Acknowledgements.

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Figure I; to Dr. Cotter Harvey for allowing me to use the findings in scalenic lymph-node biopsies; and to Mr. Woodward-Smith and his colleagues from the Department of Medical Illustration, University of Sydney, for their assistance in the preparation of photomicrographs and diagrams.

References.

ANDERSON, R. J., and CHARGAFF, E. (1929-1930), "The Chemistry of the Lipoids of Tubercle Bacilli", *J. Biol. Chem.*, 85: 77.

BESNIER, E. (1889), *"Lupus pernio de la face; synovites fongueuses (scrofulo-tuberculeuses) symétriques des extrémités supérieures"*, *Ann. derm. Syph.* (Paris), 10: 333; cited by Refvem, O, *loco citato.*

BIEBL (1938), *"Die lipophage Erkrankung des Kniegelenkes, ein bisher unbekanntes Krankheitsbild"*, *Zentralbl. Chir.*, 1: 887.

BOECK, C. (1899), "Multiple Benign Sarcoid of Skin", *J. Cutan. Genito-urin. Dis.*, 17: 543.

GHERARDI, G. J. (1950), "Localized Lymphnode Sarcoidosis Associated with Carcinoma of Bile Ducts; Report of a Case", *Arch. Path.*, 49: 163.

HARTZ, P. H., and VAN DER SAR, A. (1945), "Tuberculoid Reaction in a Retroperitoneal Neurocytoma", *Am. J. Clin. Path.*, 15: 473.

HIRSCH, E. F. (1935), "Radial Inclusion of Giant Cells", *Arch. Path.*, 20: 665.

JAQUES, W. E. (1953), "Relationship of Asteroid Bodies to Lipids", 56: 68.

LEVER, W. F. (1949), "Histopathology of the Skin", 1st edition, Lippincott, Philadelphia, London, Montreal.

LICHTMAN, A. L., McDONALD, J. R., DIXON, C. F., and MASON, F. C. (1946), "Talc Granuloma", *Surg., Gynec. & Obst.*, 83: 531.

NADEL, E. M., and ACKERMAN, L. V. (1950), "Lesions Resembling Boeck's Sarcoid in Lymphnodes Draining an Area Containing Malignant Neoplasm", *Am. J. Clin. Path.*, 20: 952.

NICKERSON, D. A. (1937), "Boeck's Sarcoid; Report of Six Cases in which Autopsies Were Made", *Arch. Path.*, 24: 19.

REFVEM, O. (1954), "The Pathogenesis of Boeck's Disease (Sarcoidosis)", *Acta med. Scandinav.*, Suppl. 294.

SABIN, F. R., DOAN, C. A., and FORKNER, C. E. (1930), "Studies on Tuberculosis", *J. Exper. Med.*, 52: Suppl. 3.

SCHAUMANN, J. (1914), *"Sur le lupus pernio. Mémoire présenté en Novembre*, 1914, *à la Société Française de Dermatologie et de Syphiligraphie pour le Prix Zambaco"*, Stockholm, 1934; cited by Refvem, O., *loco citato.*

SCHILLER, W. (1934), *"Disgerminoma und Tuberkulose"*, *Arch. f. Gynäk.*, 156: 513.

STANLEY, N. F. (1950), "Biological Properties of Polysaccharide and Lipoid Fractions from Pathogenic Strain of Aspergillus Fumigatus", *Australian J. Exper. Biol. & M. Sc.*, 28: 99.

SYMMERS, W. ST. C. (1951), "Localized Tuberculoid Granulomas Associated with Carcinoma", *Am. J. Path.*, 27: 493.

Legends to Illustrations.

FIGURE II.—Sarcoid-like lesions in lymph node of lesser omentum in a case of carcinoma of the main hepatic duct. (Hæmatoxylin and eosin stain, × 150.)

FIGURE III.—Sarcoid-like lesions in follicle of axillary lymph node in case of scirrhous carcinoma of breast. Giant-cell formation, one of which contains asteroid body. Beside the lesion strands of dense eosinophilic material are present (described by Teilum as paramyeloid). (Hæmatoxylin and eosin stain, × 150.)

FIGURE IV.—Sarcoid-like lesion in follicle of axillary lymph node in case of scirrhous carcinoma of breast. Note giant-cell formation and absence of necrosis or lymphocytic reaction. (Hæmatoxylin and eosin stain, × 300.)

FIGURE V.—Discrete sarcoid-like lesions with giant-cell reaction in axillary lymph nodes in case of *carcinoma simplex* of breast. (Hæmatoxylin and eosin stain, × 300.)

FIGURE VI.—Sarcoid-like lesion in lymph node of lesser omentum mixed with secondary formation of carcinoma of main hepatic duct. (Hæmatoxylin and eosin stain, × 150.)

FIGURE VII.—Centrally fibrosed sarcoid-like reaction with giant-cell formation and some lymphocytic reaction in liver of rat previously injected intrasplenically with 0·5 millilitre of physiological saline "extract" of scirrhous carcinoma of breast. (Hæmatoxylin and eosin stain, × 300.)

FIGURE VIII.—Sarcoid-like reaction in rat's liver. Rat previously injected intrasplenically with 0·5 millilitre of physiological saline "extract" of scirrhous carcinoma of breast. (Hæmatoxylin and eosin stain, × 300.)

FIGURE IX.—Collection of lymphocytes in periportal tract of rat's liver. Rat previously injected intrasplenically with one millilitre of physiological saline "extract" of scirrhous carcinoma of breast. (Hæmatoxylin and eosin stain, × 300.)

FIGURE X.—Pointed, slit-like space in centre of sarcoid-like lesion in rat's liver. Rat injected previously with one millilitre of physiological saline "extract" of scirrhous carcinoma of breast. (Hæmatoxylin and eosin stain, × 300.)

THE THYMUS GLAND IN MYASTHENIA GRAVIS: A CLINICO-PATHOLOGICAL REPORT.

By R. McD. ANDERSON,[1]
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IT is well known that in 20% of all cases of *myasthenia gravis* there is a neoplasm of the thymus gland (Castleman and Norris, 1949; Keynes, 1953); but the condition of the gland in the remaining patients is subject to dispute. Current text-books state that the thymus is enlarged in 50% of cases of *myasthenia gravis*, these figures being derived from the work of Bell (1917) and Norris (1936). However, Keynes (1954), in his recent examination of surgically treated patients, found that practically all non-neoplastic glands fell within the normal range of size and that many of them were small and involuted.

Sloan (1943) first drew attention to the high incidence of histological abnormality in the thymus in patients with *myasthenia gravis*. In reviewing the pathology of glands operatively removed by Blalock, he found that in seven of ten glands in which there was no neoplastic change there were lymph follicles with germinal centres. Viets (1950) found germinal centres in all of the 29 glands which he removed from patients with *myasthenia gravis*. Histologists Maximow and Bloom (1948) state that lymph follicles in the thymus are exceptional. Sloan (1943) found an occasional follicle in 14 of the thymus glands obtained from 150 subjects who had died suddenly. Castleman and Norris (1949) did not find follicles in the thymus glands of patients with no myasthenia. My own experience (20 cases) is in conformity with these findings, although only single blocks from each thymus have been examined. An increased frequency of follicles was noted by Sloan to occur in thymus glands from patients with thyrotoxicosis, acromegaly and Addison's disease.

Physiology.

No agreement has yet been reached on the physiology of *myasthenia gravis*. It is considered that in *myasthenia gravis* there is some abnormality in neuro-muscular transmission. Whether this is due to a local abnormality or to a circulating substance, or to both, is still open to question. If a circulating substance is present, then it is difficult to understand the localization of the disease to special muscle groups. On the other hand, Wilson, Obrist and Wilson (1953) have prepared an extract from the thymus glands of patients with *myasthenia gravis* and from the thymus glands of children with no signs of myasthenia, which depresses muscle contraction in nerve-muscle preparations.

The relationship of the thymus to *myasthenia gravis* is also a matter of some controversy. Keynes (1954) believes that the thymus is of prime importance. In favour of this view is the large proportion of thymomata which are associated with *myasthenia gravis*, and the preparation of an extract from the glands by Wilson *et alii* as mentioned above. On the other hand, Schwab, Leland and Boston (1953) believe that the changes in the thymus are secondary, being brought about by whatever factor gives rise to myasthenia. In accordance with this view is the fact that only 60% of patients with the disease are improved by thymectomy and that even in these the improvement frequently takes several weeks to ensue. There is also the recently reported occurrence of myasthenic states which react to "Prostigmin" and which are associated with carcinoma of the lung (Cox, 1955).

Surgery.

Surgery of the non-malignant thymus in *myasthenia gravis* has become an accepted practice in England and America, mainly as a result of the work of Keynes (1946) and of Claggett, Eaton and Bastron (1953). This type of operation was initiated by Blalock, of Baltimore, in 1936.

[1] E. H. Flack Research Scholar, 1954, 1955.

In Australia the first recorded thymectomy on a non-malignant thymus was performed by E. S. J. King in 1942 on a soldier at the military hospital at Heidelberg. This operation was carried out under local anæsthesia. At the Alfred Hospital in the period of 1952 to 1955 there have been six thymectomies performed upon patients with *myasthenia gravis*. All these operations were carried out by C. J. Officer Brown and K. Morris. In one case the gland was neoplastic and removal was impossible, whilst in the other five the glands were non-neoplastic.

Reports of Cases.

CASE I.—A, a female patient, was aged nineteen years. She was admitted to hospital on March 25, 1952. Her history was that five years previously she had first noted weakness in the legs when walking up the ramp of a city railway station. Soon after this the ocular muscles became involved and weakness became general; however, there were two partial remissions of six months each in the five years. On admission to hospital she was suffering from bronchopneumonia and poorly controlled *myasthenia gravis*. The pneumonia cleared up, but despite all forms of medical treatment her myasthenia remained refractory. Prior to thymectomy on October 24, 1952, her daily dosage of "Prostigmin" was 45 milligrammes by mouth every three hours and 2·5 milligrammes by intramuscular injection two or three times a day. After thymectomy had been performed the dosage of "Prostigmin" was gradually reduced until ten weeks after the operation its administration was stopped. No treatment has been required since then, and the patient feels better now than at any stage during the treatment of her illness. She now finds no unusual difficulty in square dancing.

On pathological examination the thymus weighed 49 grammes, and on histological examination there was very little fat present in the gland. In the medulla of the gland there were numerous lymph follicles with germinal centres (Figure I).

CASE II.—B, a male patient, aged twenty-seven years, was admitted to hospital on July 20, 1953. He had been suffering from progressive weakness in all limbs for eighteen months. Large oral doses of "Prostigmin" were not entirely satisfactory, and he was unable to sit up on waking in the morning until he took his tablets. Thymectomy was performed on July 30, 1953. Immediate post-operative progress was satisfactory, but two days after operation he developed a tension pneumothorax and died. At autopsy both lungs were found to be collapsed.

Pathological examination showed the thymus gland to be rather enlarged; its weight was 71 grammes. On microscopic examination very little fat was present and there were numerous lymph follicles in the medulla (Figure II).

CASE III.—C, a male patient, aged thirty years, was admitted to hospital on July 21, 1953. He had only a ten weeks' history of rapidly increasing weakness, starting with difficulty in chewing and swallowing, which had soon become generalized. At first oral "Prostigmin" therapy gave good relief, but on his admission to hospital three-hourly oral administration of "Prostigmin" (45 milligrammes) was only partially effective. A tumour in the anterior mediastinum was demonstrated radiologically. Operation was undertaken on August 9, 1953, but the tumour was found to be infiltrating the pericardium. Biopsy alone was carried out. Subsequently deep X-ray therapy was given and the patient improved a little; however, he contracted pneumonia and appeared to become resistant to "Prostigmin". He died on March 1, 1955, eight months after the onset of symptoms.

The initial biopsy showed a cellular tumour comprised of two cell types, thymocytes and epithelial cells, in an irregular distribution (Figure III).

At autopsy the tumour was seen to be firmly adherent to the pericardium. Histological examination revealed a good deal of fibrous tissue and cyst formation, but here and there were small areas of tumour tissue. This sort of change has been a common observation as a result of X-ray therapy to the thymus gland (Keynes, 1955).

CASE IV.—D, a female patient, was aged twenty-four years. At the time of her admission to hospital on September 17, 1954, she complained of weakness in the arms, legs and trunk, which had been getting progressively worse for three and a half years. There had been no remissions, and despite 60 milligrammes of "Prostigmin" given by mouth every three to four hours during the day her response was not complete. Prior to her admission to hospital she was unable to sit up in bed on waking in the mornings until tablets of "Prostigmin" had been taken. Thymectomy was performed on September 23, 1954, with an uneventful convalescence. Since operation she has symptomatically improved a good deal, but her "Prostigmin" requirement is almost the same as before the thymus was removed.

The thymus weighed ten grammes. Histological examination revealed a few lymph follicles with germinal centres in the medulla. There was little fat present in the gland.

CASE V.—E, a female patient, was aged forty-six years. Four and a half years before her admission to hospital on November 5, 1954, she had complained of weight loss and double vision. Exophthalmos was present. Thyrotoxicosis had been diagnosed, and radioactive iodine and deep X-ray therapy to the pituitary were given on four occasions at another hospital. During the last year she had noticed weakness commencing in the right leg, which gradually spread to her left leg and arms. Two months before her admission to hospital she began to notice difficulty in swallowing. "Prostigmin" was helpful, and at the time of her admission to hospital her daily dosage was 60 milligrammes by mouth four times a day. Thymectomy was performed on November 22, 1954. Her improvement has been mainly symptomatic.

The thymus weighed 20 grammes. There was marked involution with a good deal of fat present in the gland. The cortex and medulla appeared normal and there were no lymph follicles.

CASE VI.—F, a female patient, aged sixteen years, was admitted to hospital on June 28, 1955, with a six months' history starting with diplopia, which cleared up and was succeeded by difficulty in swallowing and speech. Associated with this was minor weakness in the arms. Despite high oral dosage of "Prostigmin" the bulbar symptoms were only partially relieved. Thymectomy was performed on July 18, 1955, with a satisfactory convalescence. At this stage, a few weeks after the operation, it is too early to judge the effect, but symptomatically she is improved and her dosage of "Prostigmin" has been reduced.

The thymus weighed 24 grammes. The medulla contained many germinal centres in lymph follicles. There was very little fat present in the gland (Figure IV).

Table I summarizes the findings in the five cases associated with non-neoplastic thymus glands.

TABLE I.

Case Number.	Sex.	Age. (Years.)	Weight of Thymus. (Grammes.)	Germinal Centres.	Fat Content.
I	Female.	19	49	++	Very little.
II	Male.	27	71	++	Very little.
IV	Female.	24	10	+	Very little.
V	Female.	46	20	−	Good deal.
VI	Female.	16	24	++	Very little.

Discussion.

The findings in this series of six cases of *myasthenia gravis* treated surgically are in conformity with those of larger overseas series. The significance of lymph follicles in the thymus is not understood; however, they are seen frequently in the thyroid gland in thyrotoxicosis. Sloan (1943) has shown that there is no generalized lymphoid hyperplasia in *myasthenia gravis*. Although the size of the thymus in *myasthenia gravis* is not increased, there appears to be a higher proportion of thymic tissue and less fat than normal.

Summary.

The findings on pathological examination of the thymus in six cases of *myasthenia gravis* treated surgically are presented. In four of five non-malignant glands there were well-marked lymph follicles with germinal centres. Lymph follicles were not seen in glands of patients without *myasthenia gravis*.

Acknowledgements.

I wish to thank Dr. L. B. Cox and Dr. John Game for permission to review their cases.

References.

BELL, E. T. (1917), "Tumor of the Thymus in Myasthenia Gravis", *J. Nerv. & Ment. Dis.*, 45:130.

CASTLEMAN, B., and NORRIS, E. (1949), "Pathology of the Thymus Gland in Myasthenia Gravis", *Medicine*, 28 : 27.

COX, L. B. (1955), to be published.

EATON, L. M., CLAGGETT, O. T., and BASTRON, J. A. (1953), "The Thymus and its Relationship to the Diseases of the Nervous System", *A. Res. Nerv. & Ment. Dis., Proc.*, 32 : 107.

KEYNES, G. (1949), "The Results of Thymectomy in Myasthenia Gravis", *Brit. M. J.*, 2 : 611.

KEYNES, G. (1954), "The Physiology of the Thymus Gland", *Brit. M. J.*, 2 : 659.

KEYNES, G. (1955), "Investigations into Thymic Diseases and Tumour Formation", *Brit. J. Surg.*, 42 : 449.

KING, E. S. J. (1943), "Thymic Hyperplasia and Myasthenia Gravis", *Australian and New Zealand J. Surg.*, 13 : 129.

MAXIMOW, A., and BLOOM, W. (1948), "A Textbook of Histology", Sixth Edition, Saunders, Philadelphia.

NORRIS, E. H. (1936), "The Thymoma and the Thymic Hyperplasia in Myasthenia Gravis with Observations on the General Pathology", *Am. J. Cancer*, 27 : 421.

SCHWAB, R. S., LELAND, C. C., and BOSTON, A. B. (1953), "Sex and Age in Myasthenia Gravis as Critical Factors in Incidence and Remission", *J.A.M.A.*, 153 : 1271.

SLOAN, H. E. (1943), "The Thymus in Myasthenia Gravis", *Surgery*, 13 : 154.

VIETS, H. R. (1950), "Thymectomy in Myasthenia Gravis", *Brit. M. J.*, 1 : 139.

WILSON, A., OBRIST, A. K., and WILSON, H. (1953), "Some Effects of Extracts of Thymus Glands Removed from Patients with Myasthenia Gravis", *Lancet*, 2 : 368.

Legends to Illustrations.

FIGURE I.—Germinal centres in lymphoid tissue from Case I (×200).

FIGURE II.—Showing lymph follicles in thymus, Case II (×110).

FIGURE III.—Thymoma, Case III, showing mixed cell appearance typical of these tumours (×760).

FIGURE IV.—Case VI: Lymphoid follicle in thymus. No Hassal's corpuscles are found in these follicles (×180).

THE MANAGEMENT OF PORTAL HYPERTENSION, WITH SPECIAL REFERENCE TO BLEEDING ŒSOPHAGEAL VARICES.[1]

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PORTAL HYPERTENSION, by which is implied increased venous pressure in the portal circulation, is commonly associated with the presence of œsophageal varices. Bleeding from these varices is by far the most important clinical phenomenon of portal hypertension. Douglass and Snell (1950) found that of 444 patients with cirrhosis of the liver 32% had a history of upper gastro-intestinal bleeding and 10% had had an episode of massive bleeding.

In this paper specific reference will be made to patients in whom there are both portal hypertension and bleeding œsophageal varices; though it is recognized that either can occur in the absence of the other. Weinberg (1949), for example, found œsophageal varices to be present in 75 out of 1189 consecutive subjects examined at autopsy and in the absence of other changes suggesting portal hypertension. This finding occurred with increased frequency in older persons. For all practical purposes, however, patients who have hæmorrhage from œsophageal varices do also have portal hypertension.

When a diagnosis of bleeding from œsophageal varices has been made, there are two particular stages of management of the patient which must be undertaken, and each involves a distinct phase of treatment. In the first stage every effort must be made to correct the effects of blood loss and to control bleeding, but later treatment is aimed at the prevention of further bleeding. In this paper both phases of treatment will be discussed after brief reference has been made to diagnosis.

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[2] Supported by a grant from the National Health and Medical Research Council and the Life Insurance Medical Research Fund of Australia and New Zealand.

Diagnosis.

Adequate management demands an adequate diagnosis; it must be established that hæmorrhage is occurring, or has occurred, from œsophageal varices, and the cause of the varices must be determined. The latter diagnosis involves a determination of the cause of the patient's portal hypertension, because this is the main factor determining the type of surgical treatment to be recommended. A study of the patient's liver function is essential for a complete assessment, but this cannot properly be carried out until after the acute bleeding episode is fully controlled.

During an acute episode of bleeding, chief reliance is placed on an adequate history and physical examination, with the addition of appropriate laboratory tests and, perhaps, X-ray examinations. However, detailed examination of the upper part of the alimentary tract is impossible. Œsophagoscopy and gastroscopy may be carried out during bleeding, but we seldom do so though such examinations have a place in some cases before an emergency laparotomy is performed.

Hæmatemeses and melæna, occurring in patients with œsophageal varices, are most probably due to rupture of the varicosed blood vessels; but it must be remembered that 10% of patients with cirrhosis of the liver and portal hypertension have upper intestinal tract ulceration (Snell, 1931). Furthermore, 90% of patients with massive bleeding from the upper part of the intestinal tract will have gastric, duodenal or stomal ulcers, and only 10% will be found to have ruptured gastric varices (Brown, Meyers, Posch and Deneen, 1950). In the cases in which diagnosis is doubtful a bromsulphthalein retention test, and X-ray examination with a barium bolus, are useful. However, balloon tamponage is the most reliable diagnostic and therapeutic test, though even here a rare exception may be encountered when bleeding is occurring from varices in the stomach.

In our experience a diagnosis of œsophageal varices is best confirmed, after bleeding has ceased, by a proper examination of the œsophagus and cardio-œsophageal region by X-ray fluoroscopy during a combined barium bolus and Valsalva manœuvre. An adequate examination of the stomach and duodenum is carried out at the same time to exclude other lesions such as ulceration, carcinoma and hiatus hernia. This X-ray examination has proved to be at least as reliable as direct examination of the œsophagus and stomach by œsophagoscopy or gastroscopy; it is obviously safer and is much more appreciated by the patient. The consistent demonstration of varices in our patients is due to the special interest and efforts of Dr. A. R. Colwell, who carries out practically all these X-ray examinations for our unit and uses a meticulous technique. We have had little experience of appearing and disappearing varices, unrelated to treatment, such as have been described by Palmer (1954), and do not think this variability to be a common cause for the failure to demonstrate varices by X-ray examination. Œsophagoscopy and gastroscopy are irregularly used, but they have their place in the diagnostic armamentarium in special circumstances. For example, one of our patients with portal hypertension had an acute bleeding episode due to hæmorrhagic gastritis, observed by gastroscopy, at a time when œsophageal varices could not be demonstrated by any means. The demonstration of gastro-œsophageal varices does not mean that a given bleeding episode was definitely due to rupture of them, though it is highly probable; the remote possibility of bleeding from other sources must always be kept in mind. The diagnosis of the cause of the portal hypertension and associated varices is usually not difficult, at least as far as determining the site of portal venous obstruction in relation to the liver is concerned. This problem will be discussed when reference is made to definitive treatment.

The Management of an Acute Bleeding Episode.

The management of patients with bleeding œsophageal varices will be discussed under three headings, as follows: the restoration of the circulating blood volume and the control of the bleeding area, the prevention and treatment of "hepatic coma", and the prophylactic measures designed to prevent recurrences.

The Restoration of the Circulating Blood Volume and the Control of Bleeding.

There is no need to describe the methods of restoring blood volume in patients in whom bleeding is occurring, and only one aspect will be discussed. The patient should not be given so great a transfusion that the hæmoglobin concentration is restored to normal. It is sufficient to raise the level to about 10 grammes per 100 millilitres. Our patients have been treated on this basis for over five years, and we have had no occasion to alter our policy. The institution of "controlled anæmia" was based on the clinical observation of Sir Charles Blackburn, that bleeding did not begin in patients with œsophageal varices, particularly in extrahepatic portal obstruction, unless their hæmoglobin concentration was normal or nearly so. This observation has been confirmed spontaneously by some of our patients' relatives. A physiological basis for this observation may be that the greater the volume and viscosity of the blood in the portal venous bed, the greater will be the portal pressure, when other factors remain unaltered, and the greater is the likelihood of bleeding from the varices. It is of interest that Spellberg (1954) makes the same recommendation, though Sherlock (1955) does not agree.

The next important step is to control the bleeding by balloon tamponage of the œsophagus and of the cardiac end of the stomach. This technique was suggested by Preble in 1900, but it was not perfected until 1950, when Sengstaken and Blakemore described the triple-lumen double-balloon tube in current use in our unit. It is a most satisfactory device for the rapid control of bleeding, and has greatly decreased the direct mortality from bleeding œsophageal varices. None of our patients has died as the immediate effect of bleeding since we started to use balloon tamponage. The Sengstaken-Blakemore tube is fitted with a round gastric balloon, approximately one-third of the way from the lower end, which is inflatable from the upper end of the tube through its own lumen. This balloon secures the lower end of the tube and prevents both regurgitation and possible asphyxiation. Above this is an elongated sausage-shaped balloon connected to a separate lumen which will compress the œsophageal mucosa directly and stop the bleeding when it is inflated. Running through the centre of both balloons is a large-bore stomach tube with multiple holes in the distal end. This tube serves both to empty the stomach and to introduce food and fluids. The tube and its attached balloons can be passed through the nose of most patients without much difficulty, and it is left inflated *in situ* for two to three days, but seldom longer (Sherlock, 1955). There is some risk of pressure necrosis if the tube is left inflated for a long time, but this complication has perhaps been overstressed. It is most important to use this tube strictly according to the full and detailed instructions issued by the designers. These instructions are supplied by the makers with each tube.

The value of balloon tamponage lies not only in its ability to control bleeding, but also in that it enables the cessation of bleeding to be determined accurately, and if the tube is left in place deflated for a day after bleeding ceases, the early recurrence of bleeding can be recognized before much blood is lost. The advantages of early feeding need no emphasis, and we prescribe a protein-poor diet until melæna ceases. If the balloons are correctly placed and fresh blood continues to appear in the aspirate, then either bleeding is occurring from a varix in the body of the stomach, which is rare, or, much more likely, bleeding is not occurring from varices at all, and other treatment is indicated. The correct siting of the balloons can always be estimated without X-ray examination, because the gastric balloon, blown up to the right size and pressure, can be pulled up to, but not beyond, the cardia by traction on the tube at the nose.

Appropriate sedatives are administered, often together with chlorpromazine. Vitamin *K* is given parenterally to help raise prothrombin levels, which are often depressed in these patients. Most barbiturates should be used with caution, because they are metabolized in the liver. In the absence of a balloon, and in cases of minor bleeding, oral feeding is forbidden, and the foot of the bed is raised to lower the venous pressure in the œsophageal varices.

The Prevention and Treatment of "Hepatic Coma".

The prevention of "hepatic coma" is especially important in patients with cirrhosis of the liver who have hæmorrhage from œsophageal varices. Hepatic coma occurs in our experience some twenty-four to thirty-six hours after the onset of bleeding, and it is not found in patients without liver disease. Whilst the cause of hepatic coma is unknown, there are at least two factors operating in cirrhotic patients with bleeding varices, apart from overenthusiastic sedation; these are anoxia of the liver, from acute blood loss, and the effects of large amounts of blood which have escaped into the intestinal canal. Anoxia is corrected by blood transfusion and needs no further comment, but the other factor has received scant attention.

We consider that the presence of blood in the alimentary tract is of major importance in the pathogenesis of hepatic coma. Similar views have been expressed recently by Sherlock (1954). The effects of blood must clearly be the same as those of a large protein meal. This is recognized as being capable of inducing coma in some patients with cirrhosis of the liver, especially in those with an Eck fistula, even in the absence of any bleeding episode (Phillips, Schwartz, Gabuzda and Davidson, 1952). Efforts should be made to remove blood from the alimentary tract as soon as possible by the administration of a simple cathartic, such as 15 grammes of sodium sulphate, either through the tube or by mouth if the tube is not being used. There are theoretical disadvantages to the use of magnesium sulphate, since the absorbed magnesium may not be excreted by the kidneys in the presence of both hypotension and concomitant renal disease. Mild diarrhœa can be induced by administering 15% to 20% glucose solution through the tube if it is thought that the small amount of sodium absorbed may add too much to the œdema.

Protein feeding has been shown to raise the blood ammonia level in patients with cirrhosis. The blood ammonia level is also elevated in hepatic coma (Gabuzda, Phillips and Davidson, 1952), though the role of ammonia in the causation of coma is not established. So long as melæna stools are being removed, the colon should be washed out twice daily to limit the production of ammonia by bacterial action upon protein, especially in the colon. In the dog, and presumably in man, the bacterial production of ammonia in the colon can be inhibited by the administration of suitable antibiotics, and "Neomycin" appears to be the best of these (Silen, Harper, Mawdsley and Veirich, 1955) and has an action much superior to that of tetracycline or sulphasuxidine. We recommend the administration of "Neomycin", one gramme every hour for four doses, and then one gramme every four hours for a total period of not more than three days, or until melæna ceases. "Neomycin" can be combined with pthalylsulphathiazole.

The treatment of coma after massive bleeding is the same as that of coma without bleeding, with the addition of the measures already described. We administer fluids, glucose, and commonly sodium glutamate as recommended by Walshe (1953)—that is, 25 grammes as a 5% solution in glucose-water daily by the intravenous route. We have not been particularly impressed by the beneficial effects of glutamate therapy, but it certainly does no harm.

Prophylactic Measures Designed to Prevent Recurrences.

After bleeding has been controlled and when the patient has recovered, it is most important to prescribe a diet

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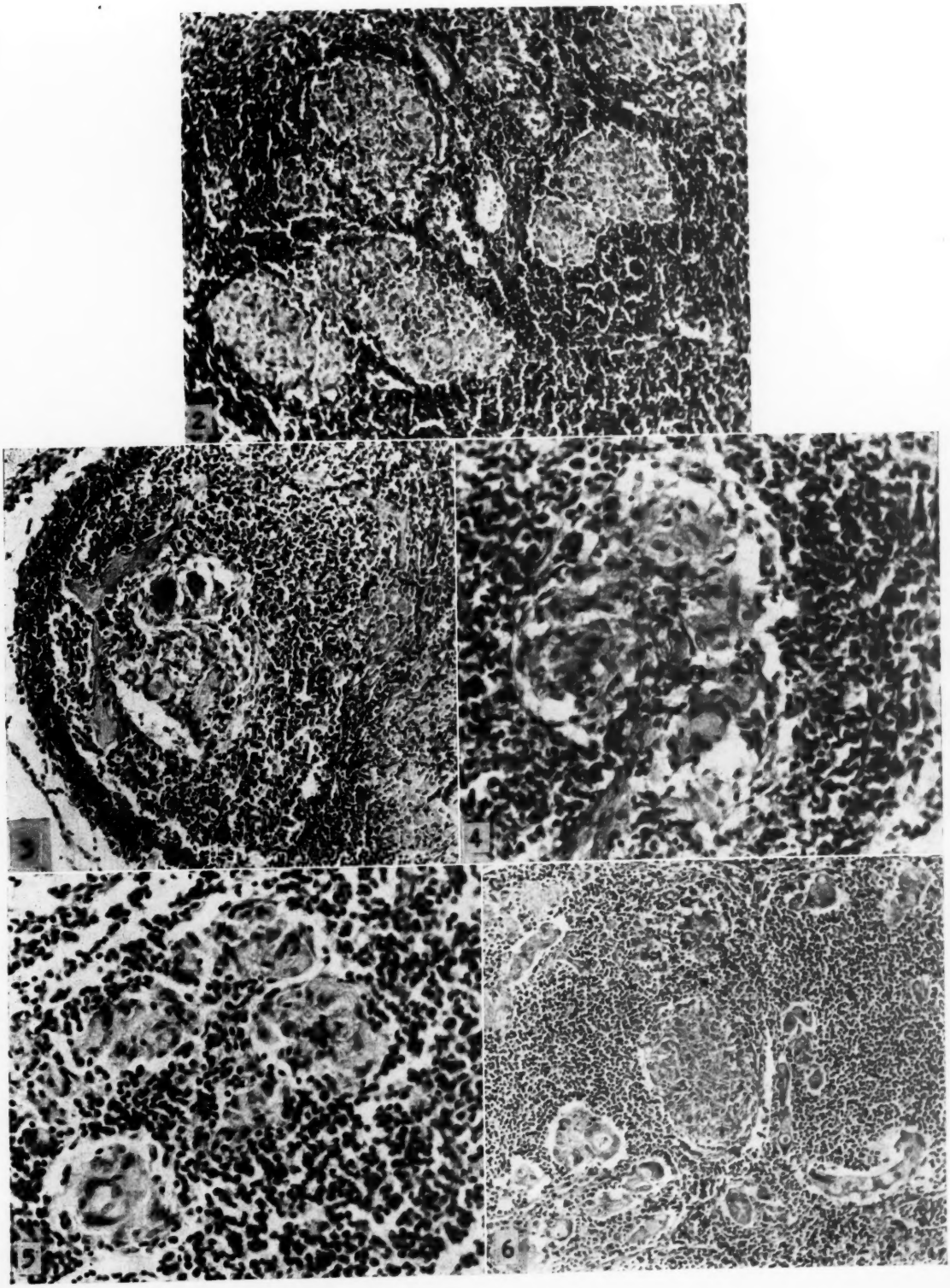

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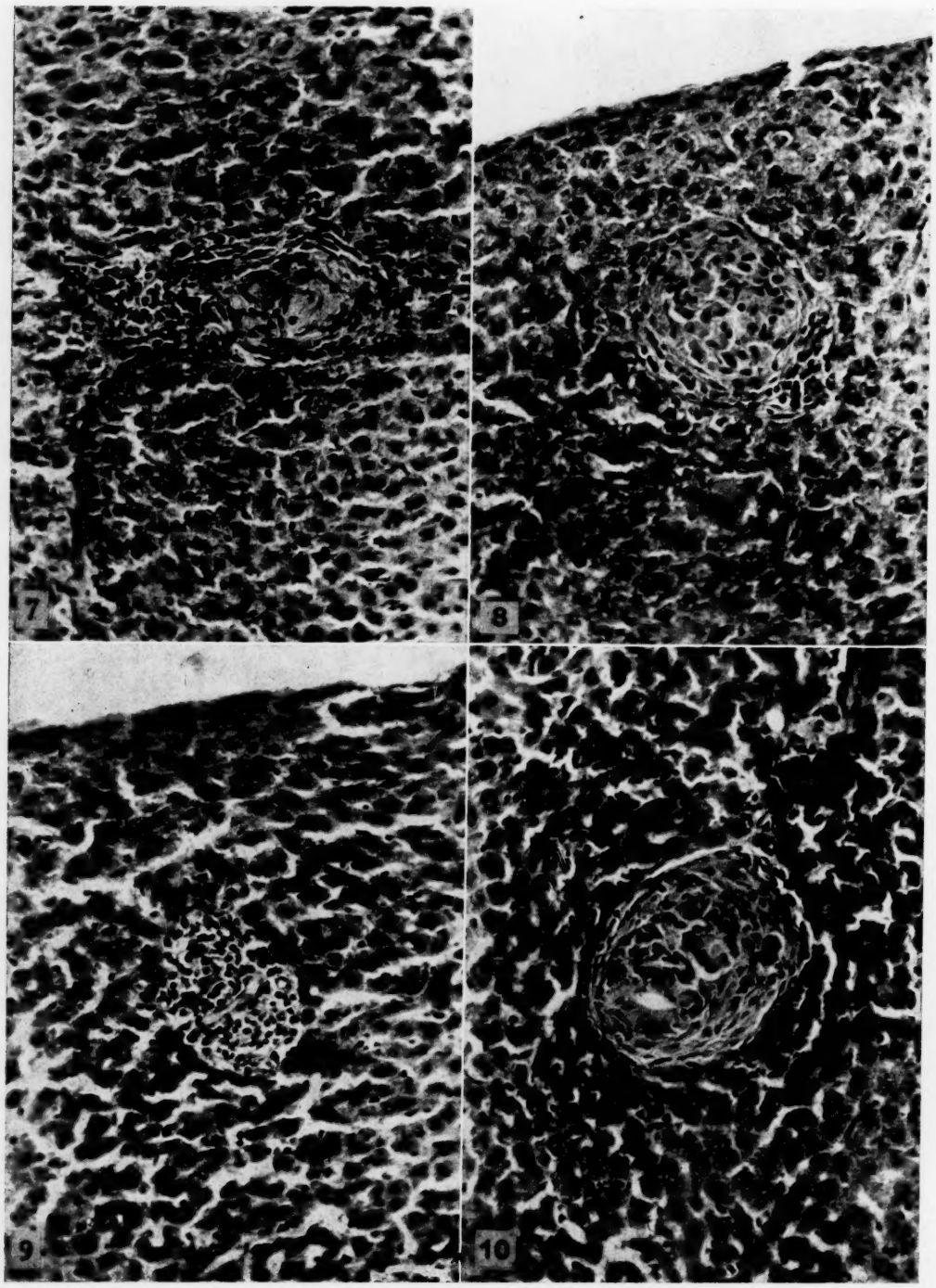

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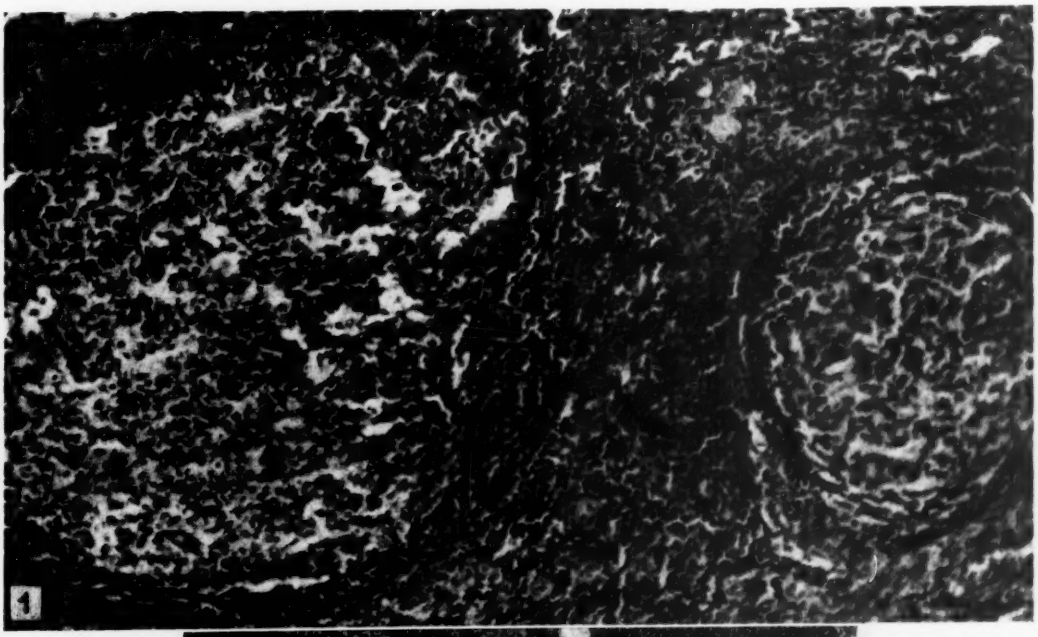
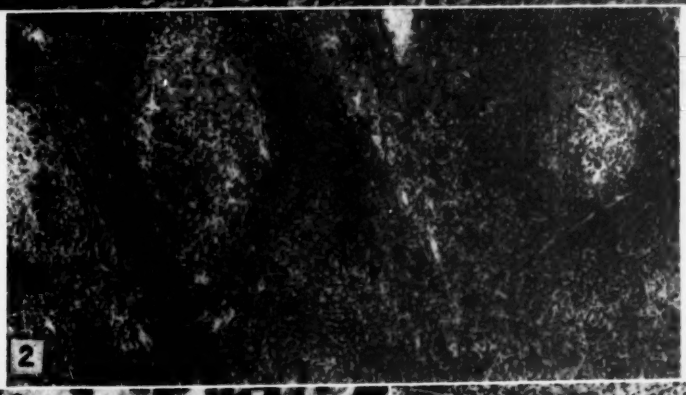
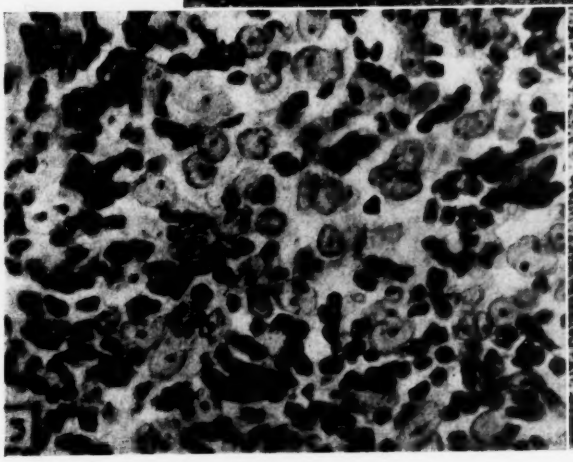
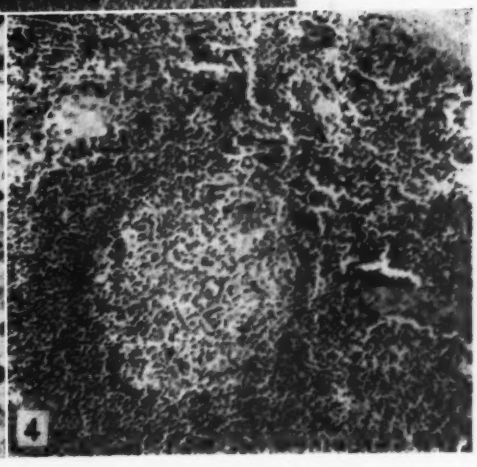
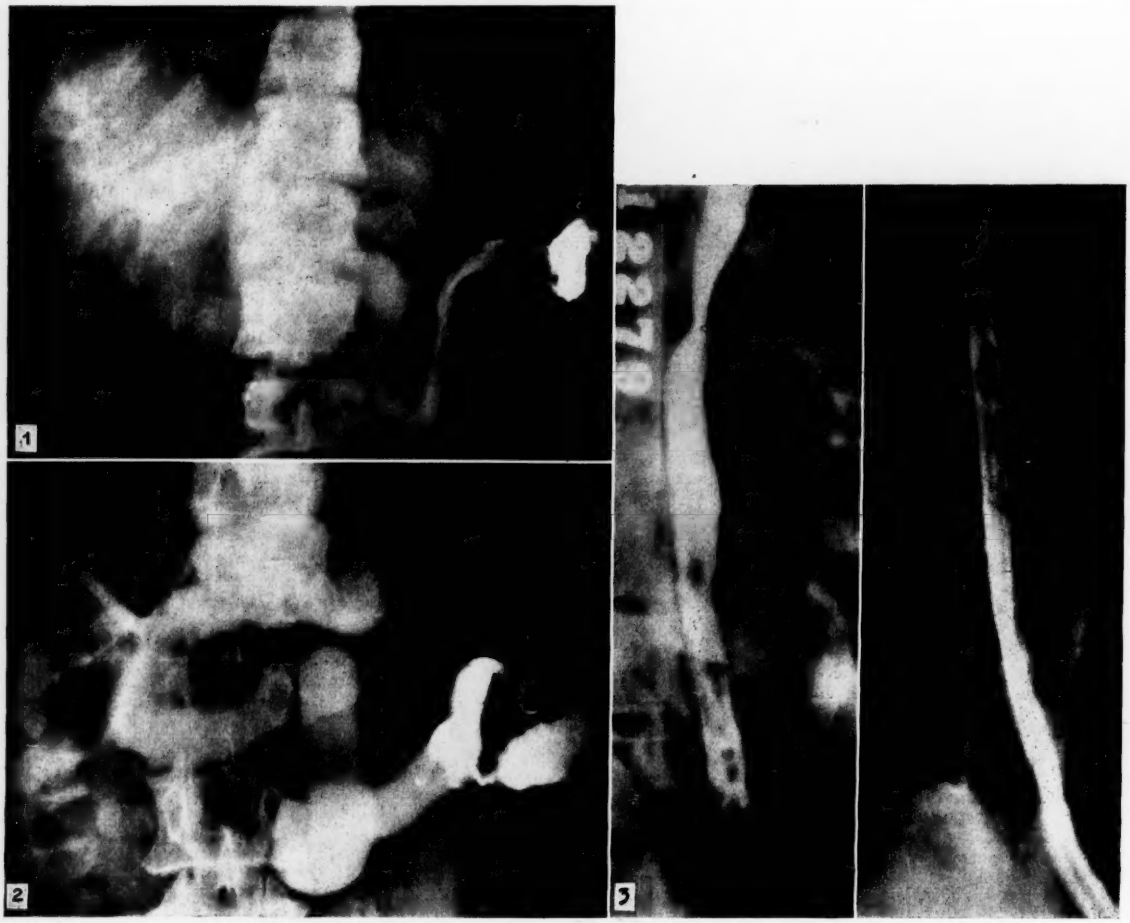








ILLUSTRATIONS TO THE ARTICLE BY C. R. BICKERTON BLACKBURN.



ILLUSTRATIONS TO THE ARTICLE BY G. H. NEILSON AND J. J. SULLIVAN.

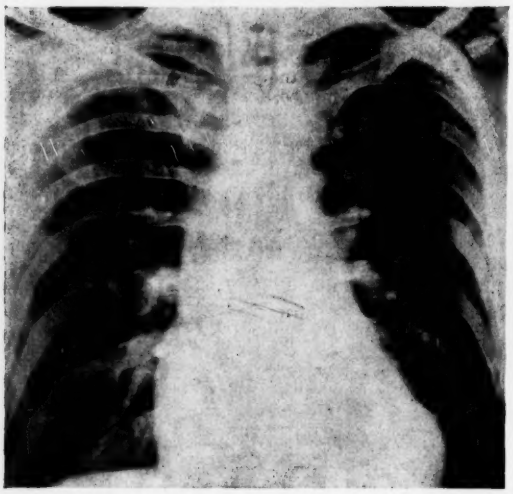


FIGURE I.

Radiograph of the chest showing broadening of mediastinal shadow, enlarged heart and congestive changes in the lung fields.

FIGURE II.

Abdominal aorta, showing cystic medionecrosis. Verhoeff's elastic tissue stain. (× 75.)

which contains nothing that could rupture a varix. One of our patients had an episode of massive bleeding after a meal during which, and for the next twenty-four hours, a fish bone was "caught in her throat". We forbid the eating of fish that may contain bones, all stews that may have bone fragments in them, such as Irish stew, and all raw hard fruits and vegetables such as green apple or cabbage. Furthermore, all substances tending to accentuate œsophagitis are interdicted—that is, strong alcohol and highly seasoned foods—and there seem to be good reasons for prescribing an alkali to limit peptic digestion of the cardio-œsophageal region. It is desirable to deal with any respiratory infections which cause much coughing, and to avoid straining at stool and the carrying of heavy weights, as these exertions tend to increase the pressure in varices in the same manner as does the Valsalva manœuvre.

In summary, the management of acute bleeding from œsophageal varices consists of the following measures: blood transfusion to restore circulating blood volume and to correct anoxia; posture, balloon tamponage and the administration of vitamin *K* to control bleeding; removal of blood from the alimentary tract and the administration of "Neomycin" to prevent hepatic coma; the giving of fluids, glucose and glutamate for coma if it occurs; and the prescription of an appropriate diet and alkali, the prevention of coughing and the interdiction of straining to minimize recurrences of bleeding.

Recurrent Bleeding.

Some patients have a series of bleeding episodes from their œsophageal varices, one episode following in the wake of another. No sooner is the balloon deflated or removed than bleeding recommences. In these cases urgent surgical treatment may be necessary. Three procedures may be considered when this emergency arises: injection of varices, ligation of varices, and definitive surgery, which is considered later. If a satisfactory condition of the patient can be obtained by tamponage, blood transfusion and general measures, we recommend the injection of varices with some sclerosing solution, such as sodium morrhuate, through an œsophagoscope. It is desirable to use tamponage after injection to limit the blood flow through the injected veins and so to hasten thrombosis. This injection procedure is difficult to perform while hæmorrhage is occurring, it is of temporary value, it is aimed only at controlling the bleeding, and it is not a definitive form of therapy.

We have not yet recommended the ligation of varices as an emergency procedure, as suggested by Linton (1953), though the operation of Crile (1950) has been carried out successfully after bleeding episodes have subsided. This is not a minor procedure, and for its performance the patient must be in good condition, especially in regard to liver function. We do not consider that the average patient with hepatic cirrhosis should have this operation, as it should be followed two weeks later by an anastomotic procedure (Linton), and these two major operations impose a severe strain on the patient.

Ligations of the hepatic artery and, possibly, the splenic artery are not in our experience brief simple laparotomies but major abdominal operations, often attended by the difficulties of exposure. If the patient can withstand this operation, he would often tolerate more effective surgical measures.

We recommend the injection or ligation of varices only for those patients with recurrent bleeding, on whom definitive operations, aimed at preventing further bleeding, cannot be carried out at the time.

Definitive Treatment.

At the present time we regard bleeding as the indication for operation, and we agree with Child (1954) that clinically significant degrees of portal hypertension do not exist in the absence of œsophageal varices which are demonstrable upon X-ray examination. By imposing the limitation of bleeding before operation, there is no doubt that we fail to offer surgical treatment to some patients who would be benefited by it. However, there is a significant mortality to operation, we are not sure of the real contraindications, and we do not know in which patients with varices bleeding will occur.

The assessment of the patient in terms of his ability to survive the stress of operation can be very difficult. At one extreme there is the patient who has a decompensated liver together with ascites, jaundice, sever hypoproteinæmia, a low prothrombin index and episodes of coma, and who is quite unfit for surgery. At the other extreme is the patient with quiescent cirrhosis, with none of the foregoing phenomena, who is usually quite fit for operation, as is the average patient with extrahepatic obstruction. In between these extremes lie most patients, and their clinical assessment may be difficult. In general, it may be said that any of the following conditions are conducive to the raising of the operative mortality: a serum albumin level of three grammes or less, intractable ascites, a low prothrombin level, strongly positive responses to flocculation tests, a bromsulphthalein retention of over 10%, a raised serum bilirubin level, a slow return of compensation after hæmorrhage, and a history of hepatic coma unassociated with bleeding. In practice one tries to assess the various relative risks of death from hæmorrhage, from operation and from hepatic failure, and one makes the decision on the particular condition of the individual patient. Our single post-operative death occurred in a patient with positive results to the flocculation tests and a low serum albumin level.

Operations designed to prevent further bleeding from œsophageal varices may be divided into those aimed at relieving portal hypertension and those aimed at removing the varicose area. Both types of operation have their place, and their particular indications depend upon several factors, of which the most important are the cause of the portal hypertension, the site of obstruction to blood flow, and the anatomy of the portal venous system.

It is usual to classify patients with portal hypertension into one of the two categories named by Whipple (1945). These are intrahepatic and extrahepatic portal obstruction, though both may be present at the same time. Intrahepatic portal obstruction is usually due to cirrhosis of the liver and is most often associated with impaired liver function, but the veins of the extrahepatic part of the portal bed are usually patent, except in the late stages of the disease. Extrahepatic obstruction is usually associated with normal or nearly normal liver function, but the portal or splenic vein, or both, is obstructed and usually there are pathological changes in the vessels themselves. Exceptions to these statements are uncommon, though they do occur, as, for example, in an aneurysm of the splenic artery compressing the splenic vein.

The role of the liver in the pathogenesis of portal hypertension must be determined prior to operation if possible. This evaluation is usually easy if an adequate history is obtained and an adequate physical examination is carried out, together with the appropriate tests of liver function. The age of onset of symptoms and the duration of bleeding episodes are important, and most of our patients with extrahepatic obstruction have given a history of bleeding from the intestinal tract from their childhood or early adult life, have had anæmia and splenomegaly and have had some evidence of hypersplenism. In spite of the occurrence of many large hæmorrhages, they present themselves in good general condition and look healthy. However, trauma, cysts, pylephlebitis from appendicitis, polycythæmia and surgical interference can also result in portal thrombosis at any age and in a variety of circumstances. On the other hand, patients with cirrhosis often have an appropriate ætiological background, they are often in poor condition, and seldom have a long history of bleeding episodes of any magnitude. The most useful liver function tests are the determination of the serum bilirubin level and the serum protein level, the flocculation tests, and the bromsulphthalein retention tests. Liver biopsy is performed in most cases if there is the slightest doubt.

The results of catheterization of the hepatic vein are very useful in determining the presence and degree of portal hypertension in patients with liver disease, especially cirrhosis, and in assessing the role of the liver in patients with portal hypertension of undetermined type. Catheterization studies also provide information on the effect of shunt operations in patients with a lowering of portal pressure. A. W. T. Edwards (1955) has published some of his results, obtained in our unit, which illustrate the value of this procedure.

Splenograms, obtained by the percutaneous injection of iodine-containing compounds into the spleen and the exposure of appropriate X-ray films (Child, O'Sullivan, Payne and McClure, 1951), are as a routine examined before operation, as by this method of examination satisfactory visualization of the portal vein and many of its tributaries can be obtained. Examples of splenograms are shown in Figures I and II. The splenogram in Figure I, obtained from a patient with myelofibrosis and splenomegaly, shows a dilated splenic vein and portal veins extending out into the liver. These findings were confirmed at operation. The splenogram in Figure II, obtained from a patient with obstruction in his portal vein, shows an enormous splenic vein and obvious obstruction of the portal vein. A diagnosis of extrahepatic obstruction of the portal vein, with a splenic vein suitable for anastomosis, was confirmed at operation.

There are only two veno-venous shunts that are regularly carried out for the relief of portal hypertension, and these are porta-caval and spleno-renal anastomoses. Other operations, such as Talma-Morison omentopexy, ligation of the hepatic artery (Reinhoff, 1951), packing of the posterior mediastinum (Som and Garlock, 1947), ligation of the splenic artery, simple splenectomy, and venous anastomoses, by the use of mesenteric or other veins, are seldom employed, because they have not been proved to be of significant value.

In our unit spleno-renal anastomoses have been performed for patients both with intrahepatic and with extrahepatic portal obstruction. The operations were performed at first by Dr. F. H. Mills, but are now performed by Professor J. I. Loewenthal, and the results have been very satisfactory. There has been no operative death, there is no evidence that any shunt has closed, and no further bleeding has occurred. In Figure III the skiagrams of the barium-filled œsophagus of one of our patients taken during the Valsalva manœuvre have been reproduced to show the presence of varices before spleno-renal shunt (left) and afterwards (right) when they could no longer be demonstrated. All the shunts have been end-to-side spleno-renal venous anastomoses with coincident splenectomy but conservation of the left kidney; porta-caval anastomoses were impossible, because of cavernomatous transformation of the portal vein, in all the patients in whom spleno-renal shunts were not possible. Surgical treatment by simple splenectomy should be reserved for those patients who have obstruction of their splenic vein or who have no splenic vein suitable for anastomosis to the left renal vein, and in whom a porta-caval anastomosis is impracticable. It is most important to avoid splenectomy unless one is prepared to perform a shunt operation at the same time.

In order to assist thrombosis and, perhaps, make it more extensive, varices should be injected as soon as possible after shunt operations. Ligation of varices should not be carried out in association with an anastomotic operation, because it should be unnecessary and it would make any subsequent œsophago-gastrectomy more difficult.

There is a group of patients who present very difficult problems, because they have had a splenectomy, but have portal vein obstruction, either primary or secondary to the thrombosis of a shunt. There is little treatment which can be recommended to these patients, with the exception of excision of the varix-bearing area, the lower end of the œsophagus, with partial or complete gastrectomy. At the present time we recommend œsophago-gastrectomy with partial gastrectomy, and have had reasonably good results, but total gastrectomy has also been carried out with success. We are inclining to the view that total gastrectomy may be the better operation, but there are particular problems relating to gastric replacement. Dr. F. W. Niesche, who carries out all the œsophago-gastric operations on our patients, has used the *Roux-en-Y* anastomosis. However, there appear to be advantages in the use of a segment of colon to replace the stomach, since one of our patients died from rupture of a jejunal varix which was situated close to the jejuno-œsophageal junction.

It is highly desirable to devise some means of preventing bleeding from varices in this last group of patients other than by extensive œsophago-gastric resection, particularly as most of those examined by us are adolescents or young adults. Child (1954) considers that patients will be free from catastrophic hæmorrhage if their portal pressures are reduced to 30 centimetres of saline or less; in other words, it is not essential to reduce the portal pressure to normal. Milnes Walker (1954) concluded that a venous shunt must be large to function satisfactorily, that it must be constructed near the site of obstruction, and that the only suitable veins on the portal side were the splenic and the portal veins. In some patients there is another large vein not too distant from an obstructed portal vein, and this is the coronary vein of the stomach. It is suggested that an anastomosis between this vein and an intrahepatic branch of the left hepatic veins or the left portal vein, or even a left hepatic vein itself, might be practicable and beneficial. Removal of some of the left lobe of the liver should not be disastrous for patients with extrahepatic obstruction and a normal liver, and should present little surgical difficulty. This anastomosis may not reduce the portal pressure to normal, but it may lower it to below 30 centimetres of saline and so prevent major bleeding. Should this be impossible, a modified type of Talma-Morison procedure could be performed between the mesentery or gastro-hepatic omentum and the liver surface, after both have been stripped of peritoneum.

The best definitive surgical measures to lower portal hypertension are spleno-renal or porta-caval veno-venous anastomoses. Partial œsophagectomy with partial or complete gastrectomy may be the only procedure available for patients in whom anastomoses cannot be carried out or have failed. It is suggested that a portal-hepatic anastomosis could be carried out between the gastric or mesenteric tributaries of the portal vein and the hepatic veins.

Conclusions.

The management of portal hypertension and bleeding œsophageal varices is medical in the first instance in most cases, but surgical measures are needed to lower portal hypertension. Treatment designed to stop bleeding is satisfactory if balloon tamponage is used, and the incidence of hepatic coma can be lessened if blood is removed from the intestinal tract. Most patients in whom an anastomosis is created between the portal and systemic venous circulations are relieved of bleeding episodes provided that the portal pressure is lowered. Spleno-renal and porta-caval anastomoses are recognized to be the most effective operations, but in some patients either these operations cannot be carried out or the anastomosis develops a thrombosis. This last group of patients can be treated by œsophago-gastric resection, but it may be possible to lower their portal pressure to a safe level by a type of portal-hepatic anastomosis.

Summary.

1. It is necessary to make an accurate diagnosis if bleeding œsophageal varices are to be treated adequately in patients with portal hypertension.

2. Balloon tamponage of the œsophageal varices is the sheet-anchor of treatment.

3. The removal of blood from the lumen of the intestinal tract and the administration of "Neomycin" are recom-

mended to lessen the incidence of hepatic coma after hæmorrhage from varices.

4. It is desirable to maintain a controlled anæmia and to prescribe a suitable non-irritating diet and an alkali as prophylactic measures against further hæmorrhage.

5. The definitive treatment of portal hypertension depends on an accurate diagnosis and an assessment of liver function, and in this regard the value of liver function tests, splenograms and hepatic catheterization has been indicated.

6. The production of veno-venous anastomoses between the portal and systemic circulations provides the best form of surgical treatment of patients with portal hypertension.

7. The treatment of patients unsuitable for the usual anastomotic operations has been discussed in terms of œsophago-gastrectomy and of the possible production of shunts between the portal and the hepatic venous systems.

References.

BROWN, J. R., MEYERS, S. G., PORSCH, J. L., and DENEEN, O. (1950), "Massive Hæmorrhage from the Upper Gastro-Intestinal Tract. A Study of 324 Cases Observed at the Detroit Receiving Hospital over a Nine Year Period", *Arch. Surg.*, 61: 767.

CHILD, C. G. (1954), "The Hepatic Circulation and Portal Hypertension", Saunders, Philadelphia and London.

CRILE, G. (1950), "Transœsophageal Ligation of Bleeding Œsophageal Varices. A Preliminary Report of Seven Cases", *Arch. Surg.*, 61: 654.

CHILD, C. G., O'SULLIVAN, W. D., PAYNE, M. A., and MCCLURE, R. D. (1951), "Portal Venography: Preliminary Report", *Radiology*, 57: 691.

DOUGLASS, B. E., and SNELL, A. M. (1950), "Portal Cirrhosis: Analysis of 444 Cases with Notes on Modern Methods of Management", *Gastroenterology*, 15: 407.

EDWARDS, A. W. T. (1955), "Studies in Portal Hypertension. Estimated Portal Pressure and Hepatic Blood Flow in Patients with Cirrhosis of the Liver and Other Disorders", M. J. AUSTRALIA, 1: 671.

GABUZDA, G. J., JUN., PHILLIPS, G. B., and DAVIDSON, C. S. (1952), "Reversible Toxic Manifestations in Patients with Cirrhosis Given Cation Exchange Resins", *New England J. Med.*, 246: 124.

LINTON, R. R. (1953), "The Emergency and Definitive Treatment of Bleeding Œsophageal Varices", *Gastroenterology*, 24: 1.

PALMER, E. D. (1954), "Portal Cirrhosis: Correlation between the Severity of Œsophageal Varices and Variations in Physical Findings", *Am. J. M. Sc.*, 227: 149.

PHILLIPS, G. B., SCHWARTZ, R., GABUZDA, G. J., JUN., and DAVIDSON, C. S. (1952), "The Syndrome of Impending Hepatic Coma in Patients with Cirrhosis of the Liver Given Certain Nitrogenous Substances", *New England J. Med.*, 247: 239.

PREBLE, R. B. (1900), quoted by Child, C. G. (1954).

REINHOFF, W. F. (1951), "Ligation of Hepatic and Splenic Arteries in the Treatment of Portal Hypertension with a Report of Six Cases: Preliminary Report", *Bull. Johns Hopkins Hosp.*, 88: 365.

SENGSTAKEN, R. W., and BLAKEMORE, A. H. (1950), "Balloon Tamponage for the Control of Hæmorrhage from Œsophageal Varices", *Ann. Surg.*, 131: 781.

SHERLOCK, S., SUMMERSKILL, W. H. J., WHITE, L. P., and PHEAR, E. A. (1954), "Portal-Systemic Encephalopathy. Neurological Complications of Liver Disease", *Lancet*, 11: 453.

SHERLOCK, S. (1955), "Diseases of the Liver and Biliary System", Blackwell Scientific Publications, London.

SILEN, W., HARPER, H. A., MAWDSLEY, D. L., and VEIRICH, W. L. (1955), "Effect of Anti-Bacterial Agents on Ammonia Production within the Intestines", *Proc. Soc. Exper. Biol. & Med.*, 88: 138.

SNELL, A. M. (1931), "Clinical Aspects of Portal Cirrhosis", *Ann. Int. Med.*, 5: 338.

SOM, M. L., and GARLOCK, J. H. (1947), "A New Approach to the Treatment of Œsophageal Varices", *J.A.M.A.*, 135: 628.

SPELLBERG, M. A. (1954), "Diseases of the Liver", Grune and Stratton, New York.

WALKER, R. MILNES (1954), "The Place of Venous Shunts in the Treatment of Portal Hypertension", *Ann. Roy. Coll. Surgeons England*, 14: 145.

WALSHE, J. M. (1953), "Effect of Glutamic Acid on the Coma of Hepatic Failure", *Lancet*, 1: 1075.

WEINBERG, T. (1949), "Observations on the Occurrence of Varices of the Œsophagus in Routine Autopsy Material", *Am. J. Clin. Path.*, 19: 554.

Reports of Cases.

DISSECTING ANEURYSM OF THE AORTA ASSOCIATED WITH MARFAN'S SYNDROME.

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THE original description by Marfan (1896) of the disease which has come to bear his name did not include abnormalities of the cardio-vascular system. Achard (1902), who introduced the term arachnodactyly, likewise emphasized exclusively the skeleto-muscular disturbances. Salle (1912) was the first to report a case in which there was a cardiac lesion—in this instance a patent *foramen ovale*. Börger (1915) also reported a case which demonstrated a similar cardiac abnormality. However, it was not until the report of Piper and Irvine-Jones (1926) that stress was laid on the frequent association of congenital heart lesions with arachnodactyly. Futcher and Southworth (1938) drew attention to an association of Marfan's syndrome with rheumatic carditis. Rados (1942) summarized 204 cases of Marfan's syndrome; sixty-three of these patients had abnormalities of the cardio-vascular system.

The features of Marfan's syndrome have been summarized by Lambie, Shellshear and Shellshear (1950), by Tung and Liebow (1952), and by Goyette and Palmer (1953). The disease is familial and congenital. The most striking abnormalities are found in the skeleto-muscular, ocular and cardio-vascular systems. In the skeleto-muscular system the chief findings are abnormal tallness and disproportionately long limbs (most pronounced in the distal portions); arachnodactyly, long thin face, dolichocephaly and high arched palate; laxity and under development of the ligaments resulting in hypermobility of the joints; spinal and thoracic deformities and muscular weakness. The commoner ocular lesions include dislocation of the lens, deformed lens, cataract, irregularity of the pupil, myopia, myosis and coloboma.

The cardio-vascular abnormalities which have been described include medionecrosis of the aorta leading to dissecting aneurysm and fusiform dilatation of that vessel; dilatation of the aortic valve ring and sinuses of Valsalva; deficiency of elastic tissue in both the aorta and the pulmonary artery; patent *foramen ovale* and auricular septal defect, patent *ductus arteriosus*, elevation of the ostia of the coronary arteries, aortic stenosis, hypoplasia of the aorta, myocardial fibrosis, and coarctation of the aorta. The problem of the incidence of rheumatic carditis in Marfan's syndrome is a complicated one. It is often not possible to decide clinically if chronic rheumatic disease is present in addition to the cardio-vascular lesions of Marfan's syndrome. Even when an autopsy is performed, confusion can be caused by the marginal thickening and nodular excrescences of the valve cusps. These occur as part of the disturbance of Marfan's syndrome and simulate rheumatic valvular lesions.

The following two cases are examples of Marfan's syndrome in which death resulted from dissecting aneurysm of the aorta.

Case I.

Clinical History.

Mr. A., a clerk, aged twenty-six years, was admitted to the Brisbane Hospital on September 17, 1953. Two hours prior to admission he had experienced a sudden onset of severe epigastric pain radiating retrosternally and into the left shoulder. Sweating and vomiting had occurred soon afterwards. The pain had increased in severity and had become of unbearable intensity. A few minutes after

the onset of the pain he had noticed a feeling of weakness in his right leg. Despite this, he had managed to walk about four hundred yards to obtain help. Three months previously he had experienced a pain of similar type, though of much less severe intensity.

Upon admission to hospital he was observed to be pale and obviously in pain. The pulse rate was 84 per minute. The blood pressure in each arm was 100 millimetres of mercury, systolic, and 40 millimetres, diastolic. The carotid, radial, femoral, posterior tibial and *dorsalis pedis* pulses were present and were equal on each side. The jugular venous pressure was normal. The apex beat was visible in the fifth left intercostal space, just beyond the mid-clavicular line. A systolic thrill was palpable over the base of the heart and over both carotid arteries. There was a harsh systolic murmur over the whole of the precordium, and a diastolic murmur of aortic type audible both at the aortic area and down the left parasternal area. At the cardiac apex a third heart sound could be heard, and a short mid-diastolic rumbling murmur was heard by one observer. Examination of the chest revealed a few crepitations at both lung bases. The liver was just palpable. The only abnormality detected in the central nervous system was some generalized weakness of the right lower limb. Examination of the fingers and toes showed arachnodactyly, and dislocation of the lens of each eye was noted.

The patient was given morphine as required for relief of pain. His condition remained the same until some thirty hours after admission to hospital, when he complained of breathlessness. On examination he was seen to be cyanosed and in respiratory distress. The blood pressure was 100 millimetres of mercury, systolic, and 45 millimetres, diastolic. The heart sounds were obscured by the breath sounds. Examination of the chest revealed crepitations at both lung bases. He failed to respond to the administration of both oxygen and morphine and died thirty-three hours after admission to hospital.

An X-ray examination of the chest three hours after admission revealed an enlarged heart and congestive changes in the lung fields. Some broadening of the mediastinal shadow was also noted (Figure I).

Autopsy Findings.

Autopsy was performed fifteen hours after death. On external examination of the body the features of arachnodactyly noted in the history were observed. The right middle finger measured 12·5 centimetres from the base of the proximal phalanx to the tip of the finger. The distance between the tip of the radial styloid and the tip of the middle finger on the right hand was 21 centimetres.

Relevant autopsy findings were chiefly in the cardiovascular system. The pericardial cavity contained approximately 50 cubic centimetres of blood-stained fluid. The left ventricular wall was 1·5 centimetres thick in its widest part. The cardiac valves were normal. The *foramen ovale* was closed. There was no elevation of the ostia of the coronary arteries. Both the interatrial septum and fat at the base of the heart were discoloured by recently extravasated blood. There was a slight dilatation of the aorta in its proximal part, with a gradual constriction to an internal diameter of 1·5 centimetres distal to the origin of the great vessels. In the descending thoracic aorta there were small raised atheromatous plaques. A ragged intimal tear, six centimetres in length, at a level one centimetre above the valve commissures, extended almost completely around the circumference of the aorta, leaving intact a segment two centimetres wide in the right posterior part of the wall. The wall of the aorta was split into two layers, at the junction of the inner two-thirds and the outer one-third. Initially this stripping involved the whole circumference of the aorta, but at the arch it spared the inferior part. Extension of the dissection into the innominate artery proceeded for a distance of one centimetre, where a reentry rupture of the intima had occurred. At the origin of the left subclavian and common carotid arteries there were two further reentry tears. The dissection continued down the aorta to the level of the bifurcation. Only the posterior half of the vessel was involved, so that those branches which arose from the anterior portion were not directly affected. The intercostal and lumbar arteries were completely separated from the inner lumen, while the left renal artery was partly separated. Reentry tears were found in both common iliac arteries, shortly after their origin. In each case these were disposed transversely on the lateral wall, the left tear being three centimetres long, the right tear five millimetres. long. Throughout the abdominal aorta the intima showed a mild degree of atherosclerosis.

The dissection had extended towards the base of the heart to below the level of the valve commissures, exposing the superior parts of the walls of the coronary arteries. The orifice of the left coronary artery was slightly distorted, the orifice of the right coronary artery was unaffected. No atheromatous deposits were found in the coronary arteries.

The only other abnormality noted was gross pulmonary œdema.

Microscopic examination of sections of the aortic wall showed the typical changes of cystic medionecrosis (Figure II). There was no myocardial fibrosis.

Case II.

Clinical History.

B., a male patient, aged twenty-four years, who worked as a house remover, was admitted to the Brisbane Hospital on May 31, 1954.

At the age of eight years he had been suspected of having rheumatic fever, but at a medical examination some months later he had been told that his heart was normal. He had been the 100 yards' distance running champion of his school at the age of thirteen years. Several hours prior to his admission to hospital, while working with a crosscut saw, he had experienced a sudden onset of retrosternal pain. He described this as a sensation of something inside his chest forcing the ribs outward. It had been of sufficient severity to cause him to fall to the ground and to break out in a profuse sweat. There had been no dyspnœa. The pain had radiated to the epigastrium and then to the left shoulder and down the left arm. Approximately five minutes after the onset of the pain he had attempted to walk and found that his left leg would not support him. He had also experienced some pain in the left hip. He complained of a pounding in the right side of his head. He had worn glasses since he was aged five years.

On examination the patient was writhing with pain. The right radial and ulnar pulses were impalpable, and the right hand was cold. The blood pressure in the right arm was 20 millimetres of mercury, systolic. The left radial and ulnar pulses were palpable, and the blood pressure in the left arm was 80 millimetres of mercury, systolic, and 60 millimetres, diastolic. Pulsation could be felt in both carotid arteries, in each of which a systolic thrill was also palpable. Both femoral pulses were palpable and equal, but a systolic thrill could be felt over the right femoral artery which could not be felt over the left femoral artery. The cardiac apex beat was palpable in the fifth left intercostal space, 10 centimetres from the midsternal line. A systolic and diastolic murmur could be heard over the whole of the precordium. The diastolic murmur was of an aortic character, maximal in the third left intercostal space, close to the sternum, and of unusual loudness. Examination of the chest revealed a few crepitations at the lung bases. The only abnormalities detected on examination of the nervous system were a generalized weakness of the left lower limb, and some hyperæsthesia and hyperalgesia of that limb. The fingers were long and thin, as were the toes. The palate had a high arch, and a severe degree of myopia was present.

Large doses of morphine were administered to the patient both subcutaneously and intravenously. Despite this there was only partial relief of his pain.

Two hours after his admission to hospital, the apex beat was noted to be 12·5 centimetres from the mid-sternal line in the fifth left intercostal space. The remainder of the findings were unchanged.

Three and a half hours after his admission the left lower limb was completely paralysed and cold, but the arterial pulsations were unchanged from those noted on his admission. The blood pressure in the right arm could not be recorded. The blood pressure in the left arm was 80 millimetres of mercury, systolic, and 60 millimetres of mercury, diastolic. The respirations were rattling in character and numerous crepitations were audible at the lung bases. Neither tendon reflexes nor plantar reflex could be elicited in the left lower limb. The patient died four hours after his admission to hospital.

Autopsy Findings.

Autopsy was performed fourteen hours after death. On external examination of the subject the elongated and slender fingers and toes were observed. The right middle finger, from the base of the proximal phalanx to the tip, measured 12·5 centimetres. The right great toe was nine centimetres in length from the base of the proximal phalanx to the tip. The length of the body was five feet eleven inches. The upper partial denture, which disclosed the arching of the hard palate, was 2·2 centimetres from the plane of the lower level of the gums to the apex of the palatal arch.

The relevant autopsy findings were again chiefly in the cardio-vascular system. The pericardial sac was normal. There was considerable hypertrophy of the left ventricular muscle, which was 2·5 centimetres in its maximum width. Examination of the mitral valve showed thickening, shortening and increased rigidity of the cusps and dense calcification of the valve ring. Cross-section of the valve ring showed an oval calcified area 1·0 by 0·5 centimetre. The left atrium was not appreciably dilated. The aortic, pulmonary and tricuspid valves were normal. The ostia of the coronary arteries were normally situated. The *foramen ovale* was closed. There was an intimal tear extending from a point one centimetre above the middle of the anterior aortic cusp and running across the posterior surface of the aorta to end above the middle of the left posterior valve cusp. The over-all length of the tear was seven centimetres. The dissection extended both proximally and distally from this tear, the split occurring at the junction of the inner two-thirds and outer one-third. Proximally the dissection reached the level of the orifices of the coronary arteries. Distally the dissection proceeded along the posterior part of the aorta. The innominate artery was dissected along its length. Reentry tears occurred at the base of the common carotid and subclavian arteries. Dissection had occurred for a short distance along the left common carotid and subclavian arteries and then the blood had reentered through intimal tears. There were two reentry tears in the aortic intima. One, at the level of the left subclavian artery, was one centimetre in length. The other, which was situated at the junction of the thoracic and abdominal aorta, was 1·5 centimetres long. There was a reentry tear in the right common iliac artery, shortly after its origin.

The vessels arising from the anterior surface of the aorta were not directly affected by the dissection. But those which arose from the posterior aspect were completely separated from the lumen of the aorta. The dissection had proceeded for a short distance along both renal arteries, but these retained their continuity with the lumen of the aorta.

There were congestion and œdema of the lungs, and there was a mild degree of passive venous congestion of the liver.

Histological studies of the aortic wall revealed typical cystic medionecrosis. Examination of the mitral valve cusp showed fibrosis and vascularization of the type seen in chronic rheumatic disease. There was no myocardial fibrosis in the sections studied.

Discussion.

Abnormality of the aorta in cases of Marfan's syndrome was first described by Baer, Taussig and Oppenheimer (1943). They reported two cases of arachnodactyly with aneurysmal dilatation of the ascending aorta, and described the changes of cystic medionecrosis in the involved portion of the aorta. Etter and Glover (1943) reported a fatal case of dissecting aneurysm of the aorta in a patient suffering from Marfan's disease, but gave no histological studies of the aortic wall. Uyeyama, Kondo and Kamins (1947) described a case of Marfan's syndrome, complicated by dissecting aneurysm, in which histological study of the aortic wall showed cystic medionecrosis. Since then there have been several reports of cases in which lesions of the aortic wall have been present.

The first of these patients presented the classical Marfan's syndrome, having skeleto-muscular, cardio-vascular and ocular abnormalities, and the syndrome was readily recognized as such. The occurrence of the previous episode of chest pain, similar to the pain with which the patient presented in his final illness, possibly represented an initial intimal tear. It is of interest that in the case of dissecting aneurysm of the aorta associated with Marfan's syndrome, which was described by Etter and Glover (1943), the patient also suffered a similar attack some months before the final episode.

The second case is of particular interest because of the less florid manifestations of the syndrome which the patient displayed. With increasing awareness of the occurrence of both fusiform and dissecting aneurysms in young people, much attention has been given to the association of these conditions with Marfan's syndrome. Goyette and Palmer (1953), and Whittaker and Sheehan (1954), have shown in some cases of dissecting aneurysm in the younger age group, that other members of the family can be found to display some or all of the manifestations of Marfan's syndrome. But the patient himself bears none of the stigmata of the condition. Minor manifestations of the syndrome, which might easily escape the notice of an unsuspecting observer, may also be found in those cases of dissecting aneurysm of the aorta in young patients. Gore (1953), in a series of 32 cases of dissecting aneurysm of the aorta in patients under the age of forty years, found only three cases in which Marfan's syndrome was present. It would be of interest to know whether the families of the remaining 29 patients showed any of the stigmata of Marfan's syndrome. Tung and Liebow (1952) also drew attention to the possibility that in patients with aneurysm of the pulmonary artery, similar search for inapparent manifestations of Marfan's syndrome and familial studies might be profitable.

The second case raises, in addition, the problem of the incidence of rheumatic heart disease in cases of Marfan's syndrome. While earlier reports stressed this association, it has become evident in recent years that the true incidence of rheumatic disease in these cases is much lower than was originally estimated. However, while the clinical history of a previous episode of acute rheumatic fever in the case presented here is too indefinite to be of value, the presence of gross calcification of the mitral valve ring, and the histological changes in the mitral valve, provide decisive evidence that the condition was rheumatic.

Summary.

1. Two cases of Marfan's syndrome complicated by dissecting aneurysm of the aorta are presented.

2. The cardio-vascular complications of Marfan's syndrome are discussed.

Acknowledgements.

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References.

ANDERSON, M., and PRATT-THOMAS, H. R. (1953), "Marfan's Syndrome", *Am. Heart J.*, 46: 911.

BAER, R. W., TAUSSIG, H. B., and OPPENHEIMER, E. H. (1943), "Congenital Aneurysmal Dilatation of the Aorta", *Bull. Johns Hopkins Hosp.*, 62: 309.

ETTER, L. E., and GLOVER, L. P. (1943), "Arachnodactyly Complicated by Dislocated Lens and Death from Rupture of a Dissecting Aneurysm of the Aorta", *J.A.M.A.*, 123: 88.

FUTCHER, P. W., and SOUTHWORTH, H. (1938), "Arachnodactyly and its Medical Complications", *Arch. Int. Med.*, 61: 693.

GORE, I. (1953), "Dissecting Aneurysms of the Aorta in Persons under Forty Years of Age", *Arch. Path.*, 55: 1.

GOYETTE, E. M., and PALMER, P. W. (1953), "Cardiovascular Lesions in Arachnodactyly", *Circulation*, 7: 373.

HALLIDAY, J. C., and LAWES, F. A. (1944), "Marfan's Syndrome: Arachnodactyly with Dislocation of the Crystalline Lens. Report of Three Cases in One Family", M. J. AUSTRALIA, 1: 465.

INGRAM, W. W., and INGLIS, K. (1931), "Arachnodactyly Associated with Muscular Hypotonia", M. J. AUSTRALIA, 2: 238.

LAMBIE, C. G., SHELLSHEAR, K. E., and SHELLSHEAR, J. L. (1950), "Arachnodactyly or Marfan's Syndrome". M. J. AUSTRALIA, 1: 213.

MARVEL, R. J., and GENOVESE, P. D. (1951), "Cardiovascular Disease in Marfan's Syndrome", *Am. Heart J.*, 42: 814.

PARKER, A. S., and HARE, H. F. (1945), "Arachnodactyly", *Radiology*, 45: 220.

PIPER, R. K., and IRVINE-JONES, E. (1926), "Arachnodactylia and its Association with Congenital Heart Disease", *Am. J. Dis. Child.*, 31: 832.

RADOS, A. (1942), "Marfan's Syndrome (Arachnodactyly Coupled with Dislocations of the Lens)", *Arch. Ophth.*, 27: 477.

ROSS, L. J. (1949), "Arachnodactyly", *Am. J. Dis. Child.*, 78: 417.

SLOPER, J. C., and STOREY, G. (1953), "Aneurysms of the Ascending Aorta due to Medial Degeneration Associated with Arachnodactyly (Marfan's Disease)", *J. Clin. Path.*, 6: 299.

TOBIN, J. R., BAY, E. B., and HUMPHREYS, E. M. (1947), "Marfan's Syndrome in the Adult", *Arch. Int. Med.*, 80: 475.

TUNG, H. L., and LIEBOW, A. A. (1952), "Marfan's Syndrome; Observations at Necropsy; With Special Reference to Medionecrosis of Great Vessels", *Lab. Invest.*, 1: 382.

UYEYAMA, H., KONDO, B., and KAMINS, M. (1947), "Arachnodactylia and Cardiovascular Disease. Report of an Autopsied Case with a Summary of Previously Autopsied Cases", *Am. Heart J.*, 34: 580.

WHITFIELD, A. G. W., ARNOTT, W. M., and STAFFORD, J. L. (1951), "Myocarditis and Aortic Hypoplasia in Arachnodactyly", *Lancet*, 1: 1387.

WHITTAKER, S. R. F., and SHEEHAN, J. D. (1954), "Dissecting Aortic Aneurysm in Marfan's Syndrome", *Lancet*, 2: 791.

Reviews.

Modern Actinotherapy: A Review of the Literature, Giving an Outline of Indications and Technique. Compiled by Raymond H. Beckett, B.A., with preface by Francis Bach, M.A., D.M. (Oxford), D.Phys.Med.; 1955. London: William Heinemann (Medical Books), Limited. 7¼" × 5", pp. 169, with 24 illustrations. Price: 17s. 6d.

THIS modest little book, clearly written by an enthusiast in this specialized field of physical medicine, is mainly a review of the literature in this field. It does not claim to be a text-book, but within its small compass it contains a mass of information that would prove valuable to the specialist in physical medicine, to the physiotherapist, and to those medical men with an interest in this work.

The author commences with an historical survey, and then deals with the physical and physiological factors involved. There is an excellent chapter on technique and dosage, followed by two entire chapters devoted to the use of actinotherapy in dermatology. Other chapters deal more briefly with its use in the rheumatic diseases, pædiatrics and tuberculosis, with a final brief chapter on miscellaneous conditions.

Considering the size of the book, there are few omissions, but two notable ones are the part played by male sex hormones in the production of pigmentation and the risk of photosensitivity produced by gold injections. On the other hand, perhaps undue prominence is given to the place of actinotherapy in the rheumatic diseases.

This remains, however, an admirable little book, containing in a small space much information, the value of which is enhanced by an excellent bibliography. It must surely be regarded as excellent value for the modest outlay involved.

Obstetrics. By J. P. Greenhill, M.D.; Eleventh Edition; 1955. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 10" × 7", pp. 1096, with 910 illustrations, 144 in colour. Price: £7.

THE eleventh edition of Greenhill's "Obstetrics" appears only four years after the previous one. The high standard set in earlier years has been maintained. This text-book succeeds in giving a complete account of obstetrics; a conservative type of management is rightly advocated, yet modern views and concepts are fully discussed; an example of the latter is the inclusion of afibrinogensemia associated with certain cases of ante-partum and post-partum hæmorrhage.

Commencing with ovulation and conception and the physiology and conduct of pregnancy, the author considers labour and the puerperium; next come pathological conditions and their management, and finally operative obstetrics is discussed in a separate section. This last section calls forth two minor criticisms; one concerns the paragraph on forceps delivery in brow presentation; surely this would be better deleted; the other criticism is directed to the routine use of a pack in the uterus at Cæsarean section.

To sum up, this is an excellent text-book for students and a reference book for practitioners. Its teaching is sound and it gives a comprehensive account of the physiology of reproduction and of the pathological conditions which may affect the mother and fœtus.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Sex Problems and Personal Relationships", by E. Parkinson Smith and A. Graham Ikin, M.A., M.Sc.; 1956. London: William Heinemann (Medical Books), Limited. 7¼" × 5", pp. 158. Price: 10s.

Intended to be read by the older adolescent, the young married or about to be married, and the older generation.

"First Studies in Anatomy and Physiology", by John Cairney, D.Sc., M.D., F.R.A.C.S., and John Cairney, B.Sc., M.B., Ch.B.; 1956. Christchurch, New Zealand: N. M. Peryer, Limited. 7¼" × 4¾", pp. 230, with 103 illustrations. Price: 30s.

Intended as an elementary text-book for maternity nurse trainees who are not already registered nurses.

"The Year Book of Drug Therapy (1955-1956 Year Book Series)", edited by Harry Beckman, M.D.; 1956. Chicago: The Year Book Publishers, Incorporated. 7¼" × 5", pp. 560, with 64 illustrations Price: $6.00.

One of the Practical Medicine Series of Year Books.

"The Control of Disease in the Tropics: A Handbook for Medical Practitioners", by T. H. Davey and W. P. H. Lightbody; 1956. London: H. K. Lewis and Company, Limited. 9¼" × 6¼", pp. 508, with 85 illustrations. Price: £2 7s. 6d.

Based on lectures given on the subject at the Liverpool School of Tropical Medicine.

"Progress in Clinical Obstetrics and Gynæcology", by T. L. T. Lewis, M.B., B.Chir. (Camb.), F.R.C.S. (Eng.), M.R.C.O.G.; 1956. London: J. and A. Churchill, Limited. 9" × 6", pp. 602, with 90 illustrations. Price: 55s.

The author's aim has been "to give an account of recent developments in clinical obstetrics and gynæcology".

"Essays in Biochemistry", edited by Samuel Graff; 1956. New York: John Wiley and Sons, Incorporated. London: Chapman and Hall, Limited. 6" × 9", pp. 355, with illustrations. Price: $6.50.

Written in honour of Hans Thacker Clarke on the occasion of his retirement as Professor and Chairman of the Department of Biochemistry, College of Physicians and Surgeons, Columbia University.

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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MIND AND MATTER.

IN medical writings and discussion over the past twenty years the adjective "psychosomatic" has been used with ever-increasing frequency. It is to be feared that, like so many other words, the adjective is widely used without any general agreement as to its precise meaning. One finds widely separated extremes of interpretation. G. W. Pickering,[1] for example, in discussing the psychosomatic hypothesis says that it "attributes disease to certain abnormal states of the mind". This simple definition conveys a picture of disorder on the psychic level being one of a number of causes, just as infection or trauma or vitamin deficiency are others. On the other hand, the definition given in a recent edition of Osler's text-book[2] is: "Psychosomatic medicine is that part of medicine which is concerned with an appraisal of both the emotional and the physical mechanisms involved in the disease processes of the individual patient with particular emphasis on the influence that these two factors exert on each other and on the individual as a whole." This definition embraces a much wider concept—that there are emotional and physical mechanisms involved in all cases of disease and that the psychosomatic approach attempts to evaluate both. It could be claimed that the growth of interest in "psychosomatic" medicine in recent years has been due to an increasing tendency to reject the viewpoint expressed in the first definition in favour of that contained in the second.

The enormous advances in curative and preventive medicine which stemmed from bacteriological, pathological and biochemical discoveries over the last century, very naturally fostered "mechanistic" ideas as to the cause of disease. The body could be regarded as a machine, albeit very complicated: disease was due to some fault in some part of the mechanism: diagnosis consisted of identifying the faulty part: treatment was an attempt to correct or prevent breakdown. The application of such principles has paid great dividends, particularly in the field of acute disease and especially acute infections. Infant and maternal mortality rates have been cut to a minimum, and many years have been added to the average life span.

With the elimination, to a large extent, of the acute infections and epidemic diseases, we have come face to face with the more chronic disorders, degenerative and neoplastic, whose ætiology is virtually unknown. In this situation, many men have raised the question whether a search for a single specific cause is sufficient or whether a wider approach is not required, and it is here that the psychosomatic philosophy has arisen again. The suggestion is that the individual must be considered as a whole, and that, in seeking an explanation for disease, it is necessary to consider the emotional and physical reaction to environment, and to look for a chain of interrelated factors which have led to the situation we call disease. J. A. Ryle[1] and J. C. Halliday[2] have supported these ideas in thoughtful papers which deserve study. E. B. Strauss,[3] in a Croonian Lecture, severely criticized the rigid concept of simple cause and effect and pointed out the limitations of a search for single specific causes. In his Bradshaw Lecture in 1953, Bomford gave an extremely lucid and readable account of recent changes in concepts of health and disease.

An objection which arises at once, however, is that while it may be desirable to have a wider philosophy, it is very doubtful whether much real knowledge of bodily function in part or as a whole can be gained by purely philosophical methods. Speculation is interesting, but fact can be established only by the application of the scientific method of controlled observation and measurement. We may be able to measure and assess the physical effects of the impact of one or more environmental factors on an individual, but can we measure with any accuracy the mental or emotional changes which it is suggested must play some part in the total reaction? Many consider that the attempt should be made and have so tried. Dunbar and her colleagues at Columbia have been pioneers in the field of studies of personality in different illnesses.[4] This work has produced much stimulating and provocative information, although it is open to the criticism that the classification of type of personality is too largely dependent on the subjective assessment of the observer. The work of S. Wolf and H. G. Wolff[5] and their associates at Cornell has given great impetus to the development of psychosomatic studies. The discovery that alterations in emotional states may be accompanied by changes in gastric and colonic mucosa is sufficiently impressive. Perhaps more important is their demonstration that the pharmacological action of administered drugs may be profoundly altered by the psychological or emotional reaction to the act of adminis-

[1] *Lancet* (1950), 2:81.
[2] Christian, H. A., "The Principles and Practice of Medicine" (Osler), Fourteenth Edition, 1942, Appelton Century, New York.

[1] *Lancet* (1942), 2:29.
[2] *Brit. J. Med. Psychol.* (1943), 19:367.
[3] *Lancet* (1952), 2:1.
[4] Dunbar, F. (1943), "Psychosomatic Diagnosis", Hoeber, Inc., New York.
[5] Wolf, S., and Wooliff, H. G. (1943), "Human Gastric Function", New York.

tration. The now well-known pathway from the hypothalamus through the pituitary to the endocrine glands and the profound and widespread physical changes which may result from liberated hormones provide one obvious link between cerebral function and physical state. The writings of H. Selye[1] have stimulated many workers to concentrate on this avenue of approach. In this issue of the journal, Bruce Robinson discusses and reviews possible linkages between psychic activity and physiological function and recalls attention to the vital role which emotional reactions may play in the production of many important disorders of the human race today. It is perhaps also indicative of the growing interest in the psychosomatic approach in this country that a very generous benefaction to the University of Adelaide has recently been used to create a senior research scholarship in the Department of Medicine, and that it is intended that the work undertaken will be concerned with problems raised by the concepts discussed here. Dr. B. S. Hetzel, who has been appointed to this scholarship, worked for three years at the University of Cornell Medical College and should be well equipped to continue this work.

As has often been emphasized, "psychosomatic medicine" is no new specialty or new concept. All states of illness are psychosomatic, just as all states of well-being depend on a proper equilibrium of psyche and soma. Since the mind cannot be divorced from matter, perhaps it is not unreasonable to attempt to study their relationships.

Current Comment.

FOOD PROCESSING AND THE VITAMINS.

PUBLIC consciousness of the nutritional value of foodstuffs is maximal at a time when food shortages are prevalent and when further supplies are imperilled. In wartime, encouraged by official advice, industrial and domestic preparation of food is designed to yield a diet as well balanced as possible, and with the vitamin content retained to the fullest possible extent. At the present time and in this country the opposite state of affairs exists. Many foods are relatively cheap and in unlimited supply, as might be expected in a country which is so important to primary producers. Vegetables are an exception; they are relatively costly, and at the same time form an essential part of the vitamin supply. Lines of communication are long, so that foodstuffs are frequently preserved or prepared. As it is easier to open a tin than to prepare the fresh article, the conditions conducive to a high consumption of pre-processed foodstuffs are now in existence. The final and most important stage at which nutritional values of foods may suffer is the storage and preparation in the home itself.

L. W. Mapson[2] has surveyed knowledge of the effect of processing on the vitamin content of foods, both from the published literature and from research carried out as part of the Food Investigation Organization of the Department of Scientific and Industrial Research in the United Kingdom. The objectives of modern food preservation are brought about by the suppression of microorganisms by increasing the osmotic pressure of the liquid phase of the food, by heat sterilization, by refrigeration, and by the addition of chemical preservatives. Loss of nutrients may occur by enzymic destruction, by chemical oxidation and by extraction. Ascorbic acid is particularly susceptible to loss by all three methods, and thus it is generally true in vegetables that the fate of ascorbic acid is a good objective index to that of the other nutrient materials. From vegetables, nutrients, especially ascorbic acid and carotene, may be lost during the cleansing and preparation, scalding, drying and canning or freezing. The loss during preparation is proportional to the extent of cutting and bruising with resultant enzymic oxidation. A widespread practice is to scald or "blanch" vegetables for a few minutes in steam or boiling water before the processing. This not only cleans the food and improves the culinary properties, but also destroys the enzymes which would destroy the vitamins. It has been found that frozen unscalded vegetables lose a large proportion of their vitamin content in a few months, while properly scalded vegetables lose little. Loss of nutritive content may also occur during scalding, owing to extraction and oxidation, especially if the constituent vegetable size is small. Water-soluble nutrients are best retained by high temperature, short-time scalding. Loss may also be reduced by using the same scalding fluid repeatedly for different batches of vegetables, and oxidation is minimized by the addition of sodium sulphide. Steam scalding has no advantage over that of water. Cooling after scalding should be by cold air and not by cold water. Little loss of nutrients occurs when vegetables and fruits are frozen. Loss of value during drying or canning is usually by oxidation, especially in the former process. Canned vegetables retain 60% of their water-soluble nutrients, though a third of this is in the fluid in the can.

Meat and fish lose no nutrients during freezing, but may lose a good deal, especially of the vitamin *B* group, in the drips of thawing. The biological value of dehydrated meat is retained, provided that the preparation temperature does not exceed 80° C. Cooking results in appreciable loss of thiamine, pantothenic acid, pyridoxine and folic acid. These nutrients are also partly lost during canning, according to the temperature and duration of the process. Fish probably suffers in the same way as meat; during smoking there is some loss of vitamin *A*.

Eggs lose little value during any of the various processes and various ways of preservation. Thiamine loss by 30% occurs during the spray drying.

Pasteurization of milk results in 20% loss of ascorbic acid and in 10% loss of thiamine. Drying results additionally in a 5% loss in the biological value of the protein, and spray drying is preferable to roller drying. Evaporation causes 60% loss of ascorbic acid and 50% loss of thiamine. Sweetened condensed milk suffers much less, with 15% loss of ascorbic acid combined with 10% loss of thiamine.

Further deterioration during the storage of food may occur if there is a rise of temperature, if oxygen is present, and if there is increase in the concentration of reactants. Dried foodstuffs should be stored in inert gases such as nitrogen or carbon dioxide. Inert gases also prevent rancidity in fatty foods, a process which results in destruction of fat-soluble and water-soluble vitamins, and which occurs in animal, fish and dairy products even at low temperatures. Ascorbic acid is totally lost during the storage of frozen vegetables in three to six months. Carotene is lost more slowly in frozen conditions; thiamine and flavine are more stable. Frozen fruit retains ascorbic acid comparatively well, though loss is rapid if it is kept after being thawed. Canned foods retain their vitamins very well over long periods.

In future, attention may be paid to the commercial use of other procedures of preservation. Heat treatment by high-frequency radiation results, if adequate, in the efficient maintenance of vitamins, both in processing and in storage. Ionizing radiations such as cathode rays, γ rays and X rays are efficient if carefully applied to foodstuffs initially high in vitamin content. The hazard of this type of procedure might be in the production of undesirable compounds in the treated foodstuffs.

Some loss of nutritive value is inevitable during the process of preserving food. For some particular articles the loss may be of little account, but for others, such as the vegetables, the loss of, say, ascorbic acid may be of particular importance in the absence of an alternative source. Cooking in the home is the final trial which

[1] Selye, H. (1950), "Stress", Montreal.
[2] *Brit. M. Bull.*, January, 1956.

nutrients must undergo. Frequently less cooking is required of preserved foods, so that further loss is much less than that of fresh cooked foods. A survey of the fate of ascorbic acid in fresh and variously preserved peas has shown that, when they reach the table from whatever their source, the ascorbic acid retained varies only between the comparatively narrow limits of 25% and 44%.

The preserved food industry is an important economic bridge between the flow and ebb of glut and shortage. It closes the miles between supply and demand, and permits the convenient and economical storage of foodstuffs both on an industrial and strategic scale and in the home. It is an industry with a high responsibility and great experience. Perhaps at times it is an asset much over-used. There is no real substitute for the fresh unprocessed article when this is available. It is a pity that the tin can and the ice-cold packet are no longer regarded as luxuries, but as expected necessities.

CESARE LOMBROSO.

IN recent years more and more attention has been devoted to the study of the criminal as a person. One of the first in this field was Cesare Lombroso, who was born in Verona on November 6, 1835, and died in Turin on November 18, 1909; a short appraisal of his work has been given by Benigno di Tullio.[1] Professor Tullio states that, although to Lombroso belongs the honour of having inaugurated the study of the criminal by scientific methods, and of having brought about enormous reforms in penal and prison law, nevertheless there are those who hold that his doctrines should now be abandoned, because they can no longer be upheld in the light of recent biological and psychological research. On the other hand, some authorities maintain that Lombroso's teachings, far from being out of date, are undergoing a natural process of evolution and adaptation to the results of biological and psychological research. Tullio is one of the latter group. Lombroso investigated the personality of common criminals, and first formulated the hypothesis of the atavistic nature of crime. This was prompted by his discovery, in a Calabrian brigand, of a "central occipital hollow" in place of the occipital crest; this is an anatomical characteristic of the lower animals. He concluded that "there are certain criminals who seem to be a residue of a former civilization now disappeared, and who, by their manner of thinking and feeling, call to mind the primitive man, incapable of assimilating the products of present-day civilization or of adapting himself, therefore, to the requirements of modern social life". Later he found symptoms of epileptic neurosis and psychosis in common criminals, and propounded the theory of the epileptic nature of crime. He was the first to draw attention to the fact that many criminals were epileptoid, that many suffered from epileptic or pseudo-epileptic crises, and that many criminal impulses were the result of psychic epilepsy. Finally, having observed the frequent presence in criminals of psychic anomalies and morbid processes, especially mental, he propounded the hypothesis of the degenerative and pathological nature of crime. As is the case with all new theories, the publication of Lombroso's hypotheses in his numerous books aroused both interest and controversy.

Tullio refers to the development of the psychological phase of criminal anthropology, which in turn was followed by the constitutionalistic-biotypological" phase, developed chiefly through the work done at the Roman School of Criminal Anthropology, on observations made on thousands of criminals in the prisons of Rome. He holds that on the results obtained it has been possible to state that there exist constitutional criminals, the principal cause of whose criminal acts is to be found in their particular constitution or in the physio-psychological structure of their personality. The result is generally a predisposition to crime, which is sustained by psychic processes and by various biological phenomena. Tullio believes that these findings have merely served to clarify Lombroso's hypotheses, and that workers in all countries are increasingly recognizing the existence of constitutional criminals with hypo-evolutional, neuropsychic and psychopathic characteristics.

Two other points in Lombroso's career not mentioned by Tullio give him the right to be remembered. In 1872 he made the discovery that pellagra was connected with the eating of a diet rich in maize. His interpretation of the finding was that there was a poison contained in maize, which was a foodstuff eaten by the peasants. Later investigations have shown that pellagra is a multiple deficiency disease and may occur in the absence of maize from the diet. However, maize contains very little tryptophane, an essential amino acid, and also probably an anti-vitamin which antagonizes the action of nicotinic acid, and therefore a diet containing much maize may induce pellagra.[1] In 1888 Lombroso, in his book *"L'uomo di genio"*, advanced the hypothesis that genius is "a degenerative psychosis, a sort of 'moral insanity' which may at times take the form of other mental disorders but which preserves certain distinguishing characteristics".[2] This proposition has given rise to much controversy. The fields of art, music, literature and science all provide numerous examples in which men of undoubted genius have behaved on occasion with remarkable eccentricity, so that at first glance Lombroso's startling hypothesis may appear entirely tenable. However, this is not the time or the place to embark upon an argument to determine its truth or falsity; it is sufficient to note that he advanced it.

It is evident from Tullio's concluding paragraphs that he believes that Lombroso initiated the scientific study of the criminal. He holds that from this point of departure, workers in this field on the basis of the most recent findings are becoming more and more convinced that the study of the personality of each criminal is of the utmost importance. Only if this is taken into account can the origin and dynamics of criminal acts be understood, and without it there can be no progress in rehabilitation of the criminal and in the elimination of individual and collective delinquency. It is unlikely that authorities anywhere in the world will dispute this. The paper is intended only as an apologia for the work of Cesare Lombroso; nevertheless, we could wish that more mention had been made of the enormous amount of work in the study of the criminal that has been done in countries other than Italy.

[1] *Sc. med. ital.*, October-December, 1955.

PROLONGATION OF LIFE IN CORONARY ATHEROSCLEROSIS.

OVER the past few years it has become increasingly apparent that atherosclerosis is related not only to inherited and inherent characteristics of the individual, but also to his way of life, his physical activity, his diet and the amount of psychological stress to which he is exposed and his reaction to it. Other factors, such as tobacco, may be involved. It seems not unreasonable to suppose that if any of these contributing factors could be reduced or abolished, the process of atherosclerosis could be slowed down and the expectancy of life prolonged. Unfortunately, by the time that the process is discovered, the individual is so set in his particular pattern of life that he may well choose to run the risks of his diseased arteries rather than abandon habits which have become an essential part of his existence. Tobacco and alcohol are dear to the individual, and it is unusual to find a man who will cheerfully abandon either. Likewise the fat and middle-aged and weary are with difficulty cajoled into taking regular exercise on theoretical considerations. The responsibilities of middle age are high, and stress and emotional tension are the inevitable outcome of a highly complex social pattern. The process of atherosclerosis is progressive, and if the cycle is to be broken, the basic metabolic abnormality must be dealt with. Studies in European nations during war-time and in countries where

[1] Garland, H. G., and Phillips, W. (editors) (1953), "Medicine", Macmillan, 1: 765.

[2] Cleckley, H. (1950), "The Mask of Sanity", 2nd Edition, 328.

the standard of nutrition is low have shown that under conditions of a poor diet and in persons of low socio-economic status the incidence of atherosclerosis and of its fatal complications is surprisingly low. In countries where diet varies only at the whim and appetite of the consumer, the incidence of atherosclerosis is very high. Of all foodstuffs, fat is the most expensive, and the proportion in which it regularly forms the diet of a community is accompanied by a proportionate incidence of atherosclerosis in members of that community. Some workers have found that the level of blood lipids tends to be high in obese persons, especially during the time of rapid weight gain. Studies have shown that atherosclerosis may be produced by a maintained high serum lipid level, and the pathological process may be induced by a diet rich in fat and cholesterol and deficient in sulpha-amino acids, methionine and cystine. The actual process is not fully understood, and it may well be that an underlying abnormality of enzyme and hormone action is responsible for the aberration of metabolism which leads to the formation of atherosclerosis. It would, then, seem logical to suppose that if obesity could be reduced, and if fat could be omitted from the diet, then at least the process might be halted despite the continuing play of extraneous factors. In these circumstances, if degeneration was not too far advanced, the secondary complications might be permanently delayed. Accordingly, a trial of the control of diet and its influence on the progress of atherosclerosis has been carried out by L. M. Morrison.[1] The author states that in American adults the atherosclerotic process is already developed when the individual is in his fourth decade of life. The group of patients studied numbered 100. Each had a proven history of coronary thrombosis with infarction of the myocardium, without any complicating metabolic disturbance. Half of the patients were not restricted in diet, and the other half were limited to a dietary fat intake of from 20 to 25 grammes daily. The mortality rate was taken as a reasonable criterion of the success of treatment. After three years, 15 of the uncontrolled and seven of the restricted group had died of cardiac disease. In addition, a considerable reduction in body weight, total serum lipid levels, total serum cholesterol levels, and neutral fat levels was observed in those patients whose diet fat was restricted.

The group of surviving patients was again reviewed eight years after the commencement of the trial. Fat control had been strictly maintained throughout this time. By this time 38 of the uncontrolled group and 22 of the diet-restricted group had died, mainly from cardio-vascular disease. The survival rate was 24% for the uncontrolled group and 56% for the restricted group. Non-cardiac causes of death were equal in the two groups, so that in fact the mortality from cardiac causes was even more pronounced than is apparent from these figures. The fact that body weight was reduced by 15% and then maintained at this nearer normal level in the fat-restricted group was probably an important factor in the patients' longevity.

The results of this study bear out what has long been concluded from the statistics of insurance companies. Greed is a human characteristic which is almost universal. Over-indulgence in all things exacts a heavy price in health and happiness. New food habits are not learnt easily by the old, and guidance into healthy diet is needed from early childhood. It has well been said that most people over forty years of age dig their graves with their knives and forks.

AXILLARY ODOUR.

MAN not only possesses the ability to detect the characteristic odour of other animals, but himself produces various bodily odours by which he is identified. Precisely how far these odours are obnoxious to either wild or domesticated animals is difficult to discern. Certainly above all human olfactory threshold levels the human odours are regarded with extreme distaste. The play upon the social dread of self-odour is one of the easiest of advertisement persuasions. The undressing rooms of out-patient departments and the peeling of clothing in slum casualty stations yield varieties of odour which steal into the dulling nostrils of the physician and cause him to wish for either a switch on the first cranial nerve or a longer stethoscope. The crowded tenements of a European slum, the packed transports of the great over-dressed and under-washed flood the nose of the sensitive with a mixture of pungent sensations. Basically the prevention of bodily odour is by bodily hygiene. As new wine should not be poured into "old bottles", neither should the clean body be draped in soiled clothing. Soiled outer garments are the pleasure ground of the moth and the anathema of the sensitive. For the unfortunate few, even constant washing and clean clothing do not appear to be sufficient, and even if their best friends remain silent, they may catch passing waves of their own exhalation. In this world when whole industries thrive on the conviction that man is not good enough as he is, the shops are liberally provided with deodorants which appear to be essential to the de-luxe model of *Homo sapiens*.

It has long been known that the axillary odour of man is associated with the presence of apocrine glands in those regions. The odour is, however, not a property of the apocrine perspiration itself. The odour is banished for many hours by washing, even when the apocrine perspiration is afterwards profuse. Comment has previously been made in this journal[1] on the original work of Shelley *et alii* which showed that the odour is produced by the action of skin-surface bacteria on the apocrine perspiration. Shaving of the axillary hairs and the intensive use of local bactericides, such as hexachlorophene detergents, considerably delay the change. The usual domestic body deodorants are the salts of aluminium, which act in the same way, although they are not universally tolerated.

W. B. Shelley and M. M. Cahn[2] have studied the problem of offensive axillary odour by means of topically applied antibiotic agents. Initial study concerned 50 normal male subjects, each of whom developed the typical axillary odour within eighteen hours of washing with soap and water. A trial was made of the effects in equal numbers of this group of a single application of a cream containing either five milligrammes per gramme of neomycin sulphate or 10 milligrammes per gramme of chloramphenicol. The cream base alone was used as a control at other times. The effect of this trial was to confirm that a single application of either antibiotic gave considerable relief from the odour for from one to four days. In the later trial five antibiotic preparations were used together with their control bases in a group of 10 men in each instance. In addition to the two previous creams, there were a lotion containing five milligrammes of neomycin sulphate per cubic centimetre, an ointment containing 2·5 milligrammes of neomycin hydrochloride and 0·25 milligramme of gramicidin per gramme of base, and a lotion containing together the same amounts of neomycin hydrochloride and gramicidin per cubic centimetre of base. These various preparations, or their base controls, were applied liberally twice daily; washing, shaving and other topical preparations were forbidden. Final odour appreciation was made two weeks later, eighteen hours after the final application. The results showed that the daily application of either neomycin cream or neomycin lotion was completely effective in preventing the odour during the period of treatment. This was partly to be expected, as the perspiration souring agent is probably a staphylococcus. The other three substances were not universally effective in preventing the odour, and this indicates that the neomycin must be present in at least 3·5 milligrammes per gramme of base to be universally effective. The cream bases were found to be quite effective in a few subjects, particularly the ones containing methyl paraben 0·1%. In two auxiliary studies the authors found that both chlortetracycline and menadione were quite ineffective when taken by mouth in

[1] *J.A.M.A.*, December 10, 1955.

[1] M. J. AUSTRALIA, February 27, 1954.
[2] *J.A.M.A.*, December 31, 1955.

the hope of preventing the fermentation of apocrine perspiration.

The results of these trials confirm the role of the bacteria in the formation of human bodily odour. A completely effective method of prevention is presented in the form of neomycin sulphate. Whether the use of the antibiotic in this way would ever be justifiable is open to question.

ELIMINATING THE STREPTOCOCCUS.

THE importance of the prevention of further recurrences of rheumatic fever, either by the administration of penicillin or by the daily prophylactic administration of sulphonamides, has from time to time received comment in this journal. Both penicillin and sulphonamides have proved to be effective in acting as a safeguard against streptococcal invasion of the throat. Views on the prevention of a first attack of rheumatic fever are rather more confused. Sulphonamides are still widely used for the treatment of acute pharyngeal streptococcal infections, and with considerable efficiency. However, F. J. Catanzaro *et alii*[1] have suggested that the development of acute rheumatism probably depends on the presence of living streptococci throughout convalescence. The sulphonamides are bacteriostatic, and thus differ from the antibiotics, which are bactericidal. It might, then, be expected that though the sulphonamide would, by suppressing infection, apparently cure the local condition, the onset of rheumatic states would be undisturbed. T. H. Stollerman[2] has suggested that complete eradication of the streptococci from the throat is essential in preventing the rheumatic sequelæ. Penicillin is highly effective against the group A streptococcus, and its use would thus be the safest way of preventing rheumatic fever. The other antibiotics might be expected to be equally effective. Stollerman insisted that penicillin should not be administered to every patient with a sore throat, but only to those with the well-known syndrome of sudden fever, sore throat, "beefy" redness of the pharynx and pharyngeal exudate. Several subsequent papers by different authors have confirmed the value of penicillin in its role in the treatment of the acute streptococcal infection. The point of disagreement concerns the use of sulphonamides in this stage, and some answer is given by A. J. Morris, R. Chamovitz, F. J. Catanzaro and C. H. Rammelkamp.[3] The subjects used in the trial of the effectiveness of sulphonamides were American servicemen admitted to hospital with exudative pharyngitis or tonsillitis, with no personal or family history of acute rheumatism, and with no history of untoward reaction either to penicillin or to sulphonamides. Group A streptococci were isolated from 261 patients who were treated with sulphadiazine, two grammes initially and one gramme every six hours for five days. Non-specific therapy was used in the treatment of 264 patients with similar diseases due to proven group A streptococci. Nine days after the onset of symptoms, the carrier rate of this organism in the throat of the sulphadiazine-treated patients was less than that of the control patients; but by the thirteenth day the carrier rates were again equal. This indicated that the streptococcus had been suppressed but not eliminated from some apparent non-carriers in the sulphonamide group. Streptococci were isolated from 88% of the convalescent sulphonamide group patients. Between one and thirty-seven days after the onset of throat symptoms, 14 of the sulphadiazine-treated and 11 of the control patients developed rheumatic fever. Streptococcal pharyngitis recurred in 21 of the sulphadiazine-treated and seven of the control patients; suppurative complications were less in the former group. The authors suggest that a single injection of benzathine penicillin G is the most effective means of preventing rheumatic fever in a patient with acute streptococcal pharyngitis. Other forms of penicillin and other antibiotics should be given for at least ten days.

The results of these various surveys indicate that sulphadiazine is of no value in the prevention of rheumatic fever following acute streptococcal infection. The great value of sulphonamides in prophylaxis is not in doubt.

[1] *Am. J. Med.*, December, 1954.
[2] *Am. J. Med.*, December, 1954.
[3] *J.A.M.A.*, January 14, 1956.

HOMOTRANSPLANTATION OF A KIDNEY.

MANY and various interesting questions are raised by a report of successful homotransplantation of a human kidney between identical twins. This was carried out at the Peter Bent Brigham Hospital, Boston, and is reported by J. P. Merrill, J. E. Murray, J. H. Harrison and W. R. Gold.[1] It is apparently the first successful operation of its kind to be reported. The only other successful kidney transplantation known was between dizygotic cattle twins, the transplant surviving and functioning for at least nine months. In the present case a man, aged twenty-four years, had begun to suffer from œdema and hypertension and was found to have extreme atrophy of both kidneys. Because of the steady worsening of the condition and the appearance of uræmia with other unfavourable prognostic signs, transplantation of one kidney from the patient's healthy identical twin brother was undertaken. Before this was done, in view of past failures when this type of procedure had been attempted, thorough investigations were carried out. Exhaustive examination by a geneticist established beyond reasonable doubt that the twins were monozygotic. More important than this, however, was the obtaining of what is described as "the most closely applicable evidence for antigenic similarity"—namely, the survival of transplanted skin between the two twins. This evidence having been obtained, it was decided to operate. The site selected for the implant was retroperitoneal and within the pelvis, the iliac vessels being used for an anastomosis. The total period of ischæmia of the donor kidney was eighty-two minutes. The other operative details are described by Merrill *et alii*, but need not be repeated here. The important thing is that function was quickly established in the transplanted kidney, and the patient's general condition steadily improved. Nevertheless, the raised blood pressure persisted; so, for this and other reasons, the two damaged and practically functionless kidneys were removed. The patient's blood pressure then became stabilized at lower and almost normal levels. Frequent urine examinations have since, up to the time when the report was written, shown disappearance of the evidence of urinary tract infection which was previously present; although some degree of proteinuria has persisted. The renal function of the homograft, as measured by the clearance of inulin and p-aminohippurate, has closely approximated that of its fellow, and excretion urography has shown prompt excretion of dye in good concentration. Merrill and his colleagues remark that the survival of the renal homograft for a period of twelve months with continuing good function indicates the complete absence of a rejection response by the host and demonstrates that renal transplantation is a technically feasible procedure. Unfortunately, there is no reason to believe that it will be a practical success other than for those lucky enough to have an identical twin.

Other matters raised provide much food for thought. An important finding is the fact that the transplantation of a normal kidney produced a dramatic response in relation to the evidence of malignant hypertension, even though the damaged kidneys remained, and on their removal the blood pressure became virtually normal; this may provide a useful clue in the understanding of the renal mechanism of malignant hypertension. Unanswered questions with important implications are: first, why one identical twin and not the other should develop glomerulonephritis; second, whether the kidney of the unaffected twin transplanted into the diseased recipient will be susceptible to further attacks; and third, whether the transplanted kidney in its unusual position in the pelvis, where it has a short and abnormally innervated ureter, will escape eventual infection.

[1] *J.A.M.A.*, January 28, 1956.

Abstracts from Medical Literature.

THERAPEUTICS.

Chlorpromazine Hepatitis.

H. GOLD, F. ROSENBERG AND W. CAMPBELL (*Ann. Int. Med.*, October, 1955) discuss chlorpromazine hepatitis and present the clinical history of three cases. Side effects following the use of chlorpromazine, which have been reported as minimal, consist primarily of drowsiness, dryness of the mouth, pyrosis, nasal congestion, constipation, miosis, mild hypotension, tachycardia, ataxia and urticaria. However, serious reactions, hepatic in origin, may occur early in the course of therapy with this drug. The three patients referred to in this report developed hepatitis after injection of the drug for two weeks. Each patient was over sixty years of age, and in each instance the patient recovered. Reference is made to previously reported cases, and it is considered to be difficult to estimate the incidence of jaundice in patients treated with chlorpromazine. In psychiatric patients treated by chlorpromazine in hospital, hepatitis has been reported in approximately 1%, and usually it appears in the second to the fourth weeks of treatment. The illness in the present three patients began with fever, malaise and anorexia, and was succeeded by the rapid development of jaundice, which clinically and by laboratory tests, proved to be due to intrahepatic biliary stasis and obstruction. The results of the hepatocellular function tests were normal, while both the serum cholesterol and alkaline phosphatase levels were considerably elevated. The phosphatase level remained high after the serum bilirubin and cholesterol levels had returned to normal. Liver biopsy is of value once the true ætiology of the hepatitis is suspected, and the other causes of biliary obstruction are ruled out. The pathological picture is that of cholestasis with plugging of the biliary canaliculi, and with varying degrees of cellular infiltration of the portal spaces. The most difficult problem presented by these patients is the differential diagnosis from extrahepatic biliary obstruction, and it seems desirable to postpone surgical exploration as long as possible whenever there is a history of the injection of chlorpromazine, in the hope that the clinical picture will clarify itself through subsidence of the jaundice, if it is due to the drug. Because of the hazards involved and the seriousness of the hepatitis that may result, it is important that the use of chlorpromazine be strictly limited to those conditions where its benefits outweigh the risks.

Unrestricted Activity in the Treatment of Pulmonary Tuberculosis.

A. R. ALLEN *et alii* (*Dis. Chest*, November, 1955), describing the favourable results of treating pulmonary tuberculosis with the three drugs, streptomycin, PAS and isoniazid, state that in their series of 101 patients unrestricted activity was allowed during the stay in hospital and that this led to patients being in better physical condition at the time of surgery. Full activity after discharge from hospital did not cause reactivation of the disease. The patients' cooperation in their treatment and in follow-up examinations is described as splendid.

Decontamination of Anæsthesia Apparatus.

G. L. GROSS (*Anesthesiology*, November, 1955) conducted a general bacteriological survey of anæsthetic equipment after it had been used. Cultures were taken separately from the endotracheal tube, suction catheter, pharyngeal airway, face-mask, Y-face-mask adapter, inspiratory tubing, expiratory tubing, breathing bags, soda lime and flutter valves. Growths of one or more of the following organisms were obtained from all these sites: *Staphylococcus aureus*, hæmolytic streptococci, *Neisseria catarrhalis*, *Pseudomonas æruginosa*, *Alkaligenes fæcalis*, *Bacillus subtilis*, *Staphylococcus albus*, *Klebsiella pneumoniæ* and micrococci. After the usual washing of this equipment with soapy water a repeated culture revealed much the same results. However, when a twelve-hour soak in "Zephiran" solution was carried out on such apparatus as Magill tubes there was no growth at all. This was followed by the adoption of a thorough soaking of all the apparatus in 1 : 1000 "Zephiran" solution for five minutes. A further detailed bacteriological survey of 12 cases both before and after the five-minute "Zephiran" soak was performed at incubation temperatures of 35° C. and 24° C. for periods of up to six days. There was found to be an almost complete decontamination of the apparatus except for *Alkaligenes fæcalis*, which was considered to be a contaminant of the "Zephiran" solution used in the experiment.

Denitrogenation.

W. K. HAMILTON AND D. W. EASTWOOD (*Anesthesiology*, November, 1955) used a series of non-medicated, normal volunteers to measure the rate and pattern of denitrogenation utilizing certain techniques of inducing anæsthesia. The gas used in these experiments was oxygen, and the percentage of nitrogen in the face-mask was measured by a Lilly nitrogen meter. The rate and degree of denitrogenation was determined by (i) the degree of ventilation of the alveoli, which is altered by minute volume, obstructed airway and the like; and (ii) the tension of nitrogen present in the inhaled atmosphere. This tension was measured with non-rebreathing, semi-closed and closed techniques, and the time taken to reach a proportion of 5% nitrogen in all three systems was less than three minutes, provided that the flow-rate of fresh gas was greater than four litres per minute. With a fresh gas flow of one litre per minute with a closed system, it took nine minutes to reduce the nitrogen percentage to 12. The effect of a small leak was demonstrated by pricking the face mask with a 15-gauge needle on inspiring deeply enough to empty the bag with the tail tube left open. The nitrogen concentration immediately rose to 60%. With one breath of room air the peak rise of nitrogen was 40%. These figures demonstrated the possibilities of there existing a low oxygen tension when faulty techniques were used with nitrous oxide and oxygen anæsthesia.

Cardiac Arrhythmias.

J. JACOBY *et alii* (*Anesthesiology*, November, 1955) investigated a group of 12 patients who were intubated under thiopentone, tubocurarine, nitrous oxide and oxygen anæsthesia with varying degrees of oxygen lack. Of the 85 patients whose oxygen saturation remained above 80%, 39 had a change after induction and 37 had no change after induction. Tachycardia was the most common abnormality after both induction and intubation. Bradycardia occurred in eight patients after induction and in none after intubation. Arrhythmias, mainly extrasystoles, occurred in five patients after induction and in eight patients after intubation. In the group with 60% to 80% of oxygen saturation, the main difference was one of arrhythmias, which occurred in 17% of subjects during intubation. In the group with an oxygen saturation of 50% to 60%, marked pulse changes occurred in 91% of cases. Four patients developed changes of pattern of the electrocardiogram, sensitivity of coupled beats, auricular flutter, wandering pacemaker, and depressed *S-T* segment. The authors conclude that vagal responses following intubation in well oxygenated patients are of little consequence; but in the presence of hypoxia they occur in almost all cases, and arrhythmias occur four times as frequently as in normal cases; these arrhythmias are more serious in nature. The effects of carbon dioxide producing arrhythmias was eliminated by using gas mixtures deficient in oxygen with full absorption of carbon dioxide rather than by deficient ventilation.

Diethyl Ether and Phosphate Uptake.

N. A. HULME AND J. C. KRANTZ (*Anesthesiology*, July, 1955) used rat-brain mitochondrial homogenates to determine the depressant effect of diethyl ether on the phosphate uptake. Their figures show that the uncoupling effect, within the range of surgical anæsthesia, corresponds to a 20% depression and becomes more severe with higher concentrations of ether. The effect on oxygen uptake did not follow the same depression curve as did the phosphate uptake. The authors propose that these experiments substantiate the hypothesis that one of the means by which anæsthetics exert their action is by the uncoupling of oxidative-phosphorylation.

Conservative Treatment of Perforated Peptic Ulcer.

H. TAYLOR AND R. P. WARREN (*Lancet*, April 14, 1956) state that each of 47 successive patients with perforated acuty peptic ulcers treated conservatively be aspiration has recovered without complications. Unlike patients with similar lesions treated by operative suture, patients in this group have shown practically no tendency towards subsequent chronic ulceration. Turning to the

treatment of perforated chronic ulcers, the authors state that treatment by aspiration involves a risk of death and is likely to lead to complications and to persistence of the ulcer; the reported results of operative suture are similar. Accordingly they consider that for a perforated acute peptic ulcer aspiration is the treatment of choice, whereas for a perforated chronic peptic ulcer partial gastrectomy is the treatment of choice. At the King George Hospital, Ilford, the following procedure has been adopted. In the casualty department the patient is given morphine and the stomach contents are removed. After the patient has been transferred to the ward and his acute distress has subsided, his condition is reviewed in detail, and the type of ulcer that has perforated is diagnosed. If it is concluded that this is an acute ulcer, gastric aspiration is continued as before. If the lesion is diagnosed as a chronic ulcer, and there are no contra-indications to operation, the patient is prepared for partial gastrectomy. Since the stomach is empty, there is no urgency about this, and the time of the operation can be arranged in accordance with the other responsibilities of the surgical staff—an important consideration in a busy hospital. If the patient has a perforated chronic ulcer but is too ill for partial gastrectomy, gastric aspiration is continued with special watchfulness for the complications that may arise.

Nephrosis and Prednisolone.

G. C. ARNEIL (*Lancet*, April 14, 1956) reports the treatment with prednisolone of four children suffering from nephrosis. He states that the treatment led to a reduction of albuminuria, considerable diuresis, a rise of serum albumin and α-globulin levels, a return to normal of the blood cholesterol level and the erythrocyte sedimentation rate, and striking clinical improvement in all four cases. In three cases albuminuria disappeared, and in the other one it was greatly reduced.

Prolonged Chemotherapy for Tuberculosis.

A. S. DOONEIEF *et alii* (*Arch. Int. Med.*, October, 1955) have treated 172 patients with pulmonary tuberculosis for an average of twenty-seven months with continuous chemotherapy in various combinations. Thirty-one patients also had surgical treatment. There has been relapse or exacerbation of the tuberculosis in only three patients. The authors believe that chemotherapy under close observation should be prolonged indefinitely, except in the presence of threatening side effects, or until a reliable guide to termination of chemotherapy becomes available.

Reserpine for Severe Hypertension.

R. PLATT AND H. T. N. SEARS (*Lancet*, April 14, 1956) report a study of 54 patients suffering from hypertension treated with reserpine. They conclude that the drug has a significant, and sometimes a profound, hypotensive effect in at least 40% of cases of severe hypertension; but that it is obviously wasteful and useless to continue its administration in cases in which there is no response. They have not been able to find any means by which the response may be foretold. They state that in most severe cases reserpine is probably better used in combination with other drugs. It is a dangerous drug. The immediate side effects are not usually dramatic; but it may cause severe depression, and this led to suicide in one of their cases. Administration of reserpine to women occasionally results in lactation.

NEUROLOGY AND PSYCHIATRY.

Presenile Dementia of the Jakob Type.

S. BORNSTEIN AND G. A. JERVIS (*Arch. Neurol. & Psychiat.*, December, 1955) discuss two cases of presenile dementia of the Jakob type from the clinical and pathological viewpoint. Two broad groups of presenile dementias are recognized: the first includes Alzheimer's and Pick's disease, the second comprises a small number of ill-defined conditions variously associated with names of investigators, such as Jakob, Creutzfeldt, Heindenhain, Kraepelin and others. The first patient whom the authors investigated was aged fifty-nine years and had, in addition to intellectual deterioration, neurological symptoms and signs similar to those of amyotrophic lateral sclerosis. In the second patient, neurological signs were limited to occasional muscular twitchings, dysarthria, occasional tremors and terminal inability to swallow. The course of the disease was rapid; it lasted sixteen months in the first patient and fourteen months in the second. The pathological lesions consisted of widespread degenerative lesions of the neurons with concomitant neuroglia reaction. In the two patients morphological changes were similar in type: in distribution they differed somewhat, the spinal cord and medulla bore the brunt of the lesions in one and the cortex and thalamus in the other. They suggest that this second broad group should be included in a single clinico-pathological syndrome under the term cortico-strio-spinal degeneration, first proposed by Wilson.

Marriage Counselling as Part of Medical Course.

K. E. APPEL, E. H. MUDD AND P. ROCHE (*Am. J. Psychiat.*, July, 1955) believe that there is a body of scientific data on family attitudes and behaviour sufficiently developed to communicate to medical students. They have had three years of experience and believe that the lectures were popular and appreciated. Thirteen lectures were given by members of the medical faculty and three additional sessions by ministers of religion. Some details of the course are given. Emphasis is laid on the limitations of advice and direction in the treatment process.

Increased Intracranial Pressure Without Localizing Signs.

L. H. BERG, H. L. ROSOMOFF, N. ARONSON, M. SILBERMANN AND J. L. POOL (*Arch. Neurol. & Psychiat.*, November, 1955) found that among 2000 patients in whom ventriculography was performed together with or without craniotomy, for a possible intracranial mass lesion, there were 238 with increased intracranial pressure without localizing signs. They concluded that the syndrome was more frequently produced by lateralized mass lesions, such as gliomata, meningiomata and subdural hæmatomata, than by serous meningitis or mid-line obstructions. Plain skull X-ray films and electroencephalographic studies were of some assistance in diagnosis. There was still a sizable number of patients in whom all clinical and laboratory evidence seemed to favour a diagnosis of serous meningitis or mid-line obstruction, but who were proved to have a laterally placed mass when ventriculography was performed.

Epilepsy and "Diamox".

C. T. LOMBROSO, D. T. DAVIDSON, JUNIOR, AND M. L. GROSSI-BIANCHI (*J.A.M.A.*, January 28, 1956) report the treatment of 126 epileptic patients with "Diamox", an inhibitor of carbonic anhydrase. They state that treatment extended for periods of from three months to three years. In 37% of cases seizures were reduced by at least 90%, in 17% they were reduced by 50% to 90%, and in 46% they were reduced by 50% or less. No patients were made worse, nor was any serious side effect or abnormality of blood, urine or bone encountered. The degree of improvement was not related significantly to a personal history of brain damage, a family history of epilepsy or the type of seizure. Benefit was greatest for patients who had three-per-second spike-wave discharges in the electroencephalogram. Maximum benefit occurred if the alkalosis induced by hyperventilation of the lungs resulted in maximal increase of spike-wave discharges and of "build-up" in the record. In some patients, well controlled for at least three months with the therapy, seizures later returned. Reinstitution of therapy with the drug later often reproduced the initial favourable response. The authors state that the beneficial effect on seizures may be the result of the acidosis or the dehydration induced, or of inhibition of enzyme action in the neurons. The drug may prove useful not only as an adjuvant in the therapy of epilepsy, but also as a means for further investigation of the role of acidosis and of carbonic anhydrase in the metabolism of normal and abnormal brain tissue.

Functional Localization in the Cerebellum.

W. W. CHAMBERS AND J. M. SPRAGUE (*Arch. Neurol. & Psychiat.*, December, 1955) discuss functional localization in the cerebellum. They support the concept of functional organization of the cerebellum in longitudinal, cortico-nuclear zones and are opposed to that theory which localizes function in the various lobes defined by comparative and embryological data. Their paper is based on the study of 41 cats, which were given specific chronic lesions. They bring forward further evidence to support this concept as presented by Jansen and Brodal.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on Tuesday, February 21, 1956, the assistant medical superintendent, DR. J. E. REIMER, in the chair. The principal speaker was DR. K. B. NOAD, an honorary physician of the hospital.

DR. J. E. REIMER: I should like to introduce Dr. Noad to discuss this interesting case.

Clinical History.

The patient, a thirty-two-year-old single woman, a buyer by occupation, was admitted to hospital on November 22 for investigation of pyrexia of unknown origin. She had been well until a year prior to admission, when she developed painful legs, knees and hands. This was diagnosed as arthritis, and she was given a course of liver injections. Later she collapsed and was told she had "gastric influenza" and "congestion". In August she again developed "influenza", and this recurred in October, since when she had sweated a good deal and felt hot and clammy, with palpitations, dyspnœa on effort and continual tiredness. In spite of a good appetite she lost one and a half stone in weight in the year. There was no relevant past history or family history.

On examination she was found to be a pale thin woman with spindle-shaped swellings of the interphalangeal joints, scattered areas of bronchial breathing in both lungs and medium crepitations throughout the chest. Albumin was present in the urine. Her temperature was 101° F. and her pulse rate 120 per minute. There were no other abnormalities noted.

A provisional diagnosis of bronchopneumonia and rheumatoid arthritis was made.

Chest X-ray examination showed diffuse mottling throughout both lungs which could be miliary tuberculosis, bronchiolitis or carcinoma. The heart shadow was enlarged and indicated dilatation.

The Mantoux reaction was negative at first strength and positive at second strength.

Sputum examination showed no acid-fast bacilli in four specimens examined.

Blood examination revealed a blood sedimentation rate of 36 millimetres in one hour (Westergren), a hæmoglobin value of 9·5 grammes *per centum* and a white cell count of 6700 per cubic millimetre (neutrophile cells 45%, lymphocytes 39%, monocytes 14%, basophile cells 2%).

Because of the X-ray findings a diagnosis of miliary tuberculosis was made, and treatment begun on December 1 with streptomycin, 0·75 gramme twice a day. Three days later she had headache, and there was a fleeting suggestion of neck rigidity and a positive Kernig's sign. Streptomycin dosage was increased to one gramme three times a day on December 11. She developed severe headaches, attributed to a toxic reaction to the drug, but no neck rigidity. The physical signs in the lungs were unchanged, the hæmoglobin value 10·7 grammes *per centum*, the white cell count 5750 per cubic millimetre and the blood sedimentation rate 66 millimetres in one hour. Radiologically the foci in the lungs appeared less numerous and sharper in outline. Her temperature, which had been 100° to 103° F., became normal.

During January the headaches were severe, she behaved "peculiarly" at times and was drowsy, the left knee jerk was diminished, and the possibility of tuberculous meningitis was considered. From a lumbar puncture clear fluid was obtained under pressure of 120 millimetres of fluid containing protein 50 milligrammes, chlorides 675 milligrammes and glucose 30 milligrammes per 100 millilitres, and 60 lymphocytes, 20 polymorphonuclear cells and three red cells per cubic millimetre. No organisms were seen in the fluid.

In February, treatment was commenced with intrathæcal injections of streptomycin, 200 milligrammes every second day. Next day the patient became unconscious. Two days later a polythene tube was inserted into the interpeduncular cistern through a right fronto-parietal flap and 0·25 gramme of streptomycin was injected twice daily. Her temperature, which had been normal for six weeks, rose to 103° to 104° F., and two days later she died.

Clinical Discussion.

DR. K. B. NOAD: This story concerns a young woman of thirty-two years of age who, during a year of arthropathy which was very suggestive of rheumatoid arthritis, began to run an unexplained fever, for the investigation of which she was admitted to this hospital. I should think the medical attendant was probably glad that she went to hospital. For her arthropathy and other conditions she had a course of liver injections, and she had diagnoses from time to time of "gastric influenza" and "congestion" and the like. These are usually refuges of the destitute, and she obviously needed further investigation. There is a priceless statement in the notes: "Later she collapsed and was told she had gastric influenza and congestion." I wonder what that means. Incidentally that word "collapse" is one of my *bêtes noire*. I think it is almost as obnoxious as the word "blackout". It is not only in medical parlance or in the parlance of patients that the word exists, but, of course, it is unusual to open the evening newspaper without seeing that Mrs. So-and-So collapsed when told she had won the lottery or X, the reigning sprint champion, when being beaten by the contender for the title, collapsed and complained there was a hole in the track, although none could be found at subsequent examination. I just do not know what "collapse" means. Anyway, I do not think the word "collapse" has any place in any medical history unless it can be more specifically defined.

An X-ray film taken soon after admission showed diffuse mottling of the lungs diagnosed as miliary tuberculosis, and treatment was begun on those grounds. Towards the end of the second week of her hospital stay she developed headache and some meningeal signs, though these do not appear to have been very significant. During the second month of her admission the headaches became very severe, and she developed signs of cerebral involvement, such as stupor, drowsinss, peculiar behaviour and so forth. Despite treatment she was obviously going downhill. Intrathecal therapy with streptomycin was begun, and rather a large dose was given—200 milligrammes as a first dose. Rarely is more than 100 milligrammes given, and even that has to be well diluted. Anyhow, she then became unconscious, and heroic measures were instituted; a polythene tube was put into the interpeduncular fossa, and 250 milligrammes of streptomycin were instilled twice a day. This was too much for her, and she died.

Could we see the X-ray films? Will you comment on these, Dr. Marsh?

DR. H. G. MARSH: A series of X-ray films was taken during her stay in hospital. This one was on December 1, and we see a diffuse mottling, described as a miliary mottling in the lungs. I have given some time to reading about such mottling since I heard this case was to be discussed, and I think the main point is that it is entirely non-specific. There are many causes of miliary mottling in the lungs, but in any case only a few are applicable. By December 20 the changes were a little less obvious. That is as far as I am prepared to go at this stage.

DR. NOAD: At first glance one would think that this was a polysystemic disease. There is evidence of involvement of quite a number of systems: fever, arthropathy, infiltration of the lungs, dilatation of the heart, albuminuria, anæmia and a raised sedimentation rate. A disease of vessels, such as *periarteritis nodosa*, which could account for many of these phenomena, could also account for the cerebral signs and symptoms which the patient had. It could also cause the changes in the cerebro-spinal fluid. As you see, she had slightly raised protein and a pleocytosis. Perhaps the reduction in sugar is not so easily explained, but the symptoms of drowsiness and peculiar behaviour could well be explained by vascular disease of this character. After all, *periarteritis nodosa* produces an ischæmia, and ischæmia in appropriate areas could produce those symptoms. However, such a condition is usually associated with a leucocytosis, and yet this patient had on two occasions a total leucocyte count within normal limits. It is usually also associated with hypertension; but, I must inform you, although this patient was in hospital close on three months, the record contains only one reading of blood pressure (150 millimetres of mercury, systolic, and 76 millimetres, diastolic) taken on admission. I submit we really had no information about the blood pressure. However, a polysystemic disease often does not have every feature. If we accept this diagnosis then, the arthropathy was not rheumatoid arthritis but part of a more general disease. But let us say, for the sake of argument, that she did have rheumatoid arthritis and that the raised sedimentation rate, the anæmia and the joint changes were due to this and were not associated with what follows. Then we must begin to

discuss other conditions associated with loss of weight, fever, infiltration of the lungs and meningo-encephalitis. Incidentally, talking about the many conditions causing infiltration of the lungs, rheumatoid arthritis is sometimes associated with it, though there we must stop, as a meningo-encephalitis such as this woman had is very unlikely in rheumatoid arthritis.

The causes of these other features are tuberculosis and carcinoma, torulosis, chronic sepsis of the lung with cerebritis, and perhaps we might throw in, from the radiological point of view, sarcoidosis.

To deal with tuberculosis first, I think there is considerable evidence that she did not have it. Firstly, there was the negative first strength Mantoux response. Though theoretically this does not exclude tuberculosis, in my opinion it is a very practical objection to the diagnosis. We can talk about the anergic phase, but the negative Mantoux response is a very serious objection. Her sputum was "negative", but this is not very important. Then I thought that the diagnosis of tuberculous meningitis was made on quite scanty evidence. The clinical signs were a positive Kernig sign and a "fleeting suggestion" of neck rigidity. Now, I have seen a patient with tuberculous meningitis with a positive Kernig sign and slight neck rigidity, but it was not fleeting, and it was not a fleeting suggestion. That seems to be very slight. But these patients are exceptional. Usually in tuberculous meningitis the meningeal signs are strong. So the clinical signs of tuberculous meningitis, to my mind, were scanty.

Then the cerebro-spinal fluid was not characteristic. I have had put on the board the cerebro-spinal fluid findings in the last seven cases of tuberculous meningitis which have been in this hospital. You see that, by and large, the cell counts are high, the protein level is high, the chlorides are low in amount and the sugars are variable. In this patient the chlorides were not reduced sufficiently in amount to make this diagnosis, in my opinion; the glucose level is low, but not enough to be diagnostic; and the protein level is hardly high enough. Moreover, this seems to be a meningo-encephalitis rather than a meningitis. The response to streptomycin, too, despite very large doses, was poor. Certainly in the second X-ray film of the chest, as we have seen, the foci were obviously less and clearer cut, but that might be explained by the disappearance of œdema due to rest. Perhaps, too, the streptomycin had a non-specific effect on some bacteria other than tubercle bacilli in those lesions. So I am excluding tuberculosis.

To deal with the other conditions, torulosis is a rare condition, but we have had quite an experience of it in this hospital. The common radiological lesion in torulosis is a single opacity, the toruloma. However, Cox and Tolhurst in their monograph report more than one instance of miliary torulosis. This, together with the cerebral symptoms, would fit the diagnosis of torulosis admirably. In this condition the protein level is raised and the sugar level lowered, the cells being increased just as in this case. But I find one difficulty in applying this diagnosis, and that is the statement in the history that over a period of many months she had recurring bouts of fever. Our patients have usually been febrile, mildly so, just as Cox and Tolhurst found in most of their patients, but I have been unable to find reference to patients having recurring bouts of fever. Moreover, I think there is usually an increase in the leucocyte count in torulosis. I will leave that condition.

Carcinomatosis has to be considered both on clinical and on radiological grounds. Infiltration of the brain and lungs is quite common in carcinomatosis, and I was intrigued by the reference to the absent left knee jerk as it reminded me of a patient I had. I have a slide showing that patient's cord, which was heavily infiltrated in the lumbar region. The protein level in that patient was very high, about 2000 milligrammes per 100 millilitres. The recurrent febrile attacks are quite compatible with a diagnosis of carcinomatosis, and this association of carcinoma with fever has always interested me. It occurs particularly in primary kidney cancer, with metastases from the breast, metastases in the liver, and also with another type of growth, Ewing's sarcoma. I have even seen a patient with carcinomatosis from a primary in the breast and infiltration in the lung who had a tertian type of fever exactly like malaria. The loss of weight would, of course, accord with the diagnosis of carcinoma, but the retention of a good appetite would not. In fact the retention of appetite does not accord well with any of these conditions, except perhaps the first-mentioned arteriopathy.

From the radiological point of view I mentioned sarcoidosis, only to exclude it. It has to come into the differential diagnosis of any infiltration of the lung, but recurrent bouts of fever would be most unusual, I think, as would the meningo-encephalitis. To come to the last condition, it is chronic sepsis of the lung. When I was a student, I was always taught that, when confronted with a case of unexplained fever, there were four conditions to be thought of—tuberculosis, malaria, typhoid fever and sepsis. Well, we have considered tuberculosis; typhoid we do not see very often now; malaria I think we can exclude; and so we come to consider sepsis. This patient was admitted for the investigation of pyrexia of unknown origin, and there is a lot in the record to suggest a background of infection. There were those diagnoses of influenza, which have rather a respiratory flavour about them. She sweated, felt hot and clammy, was tired and had lost weight. On admission she was found to have scattered areas of bronchial breathing with crepitations throughout the lungs. This is all compatible with diffuse sepsis in the lungs. The radiologist thought fit to include bronchiolitis in his differential diagnosis. I would point out, however, that there is no mention of cough in this record. A metastatic cerebritis and abscess formation is a very common accompaniment of pulmonary sepsis as you know, and the cerebro-spinal fluid findings here would be quite in keeping with such a condition. The sharpening of the outlines of the miliary opacities in the lung could be, as I said before, due to the combined effects of rest and the non-specific, shall we call it, effects of streptomycin therapy. It was at this time that her temperature fell to normal; which could also be taken to support the diagnosis. But there is one objection to the diagnosis, and that is that the leucocyte count was within the normal range. I am told that no blood culture was made. In passing, I would say that sometimes it is very interesting in blood-stream infections that the white cell count is normal or even low. I have laboured over these diagnoses—a vascular lesion like periarteritis, torulosis, carcinomatosis, chronic respiratory sepsis and tuberculosis. To my mind, there are objections to each of them. To periarteritis there are the normal leucocyte counts and doubtful blood pressure. Torulosis I exclude because of the history of recurrent attacks of fever and the normal leucocyte counts. Carcinoma, I think, we must exclude on the grounds of the lessening and sharpening of the miliary shadows; and, if a metastasis was causing the loss of knee jerk, I would expect the protein level in the cerebro-spinal fluid to be much higher Sepsis: the normal leucocyte counts are an objection.

If I am forced to make a choice, it will have to be for the condition for which there is the least objection, and that is a polysystemic disorder such as *periarteritis nodosa* with involvement of the lung and also of the cerebrum as well.

DR. REIMER: Thank you, Dr. Noad. Would you like to comment, Dr. Calov?

DR. W. L. CALOV: I would agree with Dr. Noad's opinion that it cannot be tuberculosis—at least I do not think it can be. If it were, this is just the kind of case in which streptomycin, particularly in the big doses used here, produces a good result. I wondered about the possibility of a fungous disease, especially torulosis, but X-ray films of the chest of the ones I have seen showed just one solitary mass. That, to me, is the picture of the disease—a granuloma, with one solid lesion. The rest of the story would fit torulosis fairly well, though, as Dr. Noad says, the history is a long one. Of course, some of these other febrile disturbances might not have been associated with her main disease. However, torulosis can persist for quite a long while, so I would not think that the duration of the illness rules it out. In my opinion it is more likely that one of the other fungous diseases, like coccidioidosis, was present. I would agree with Dr. Noad that the condition is not carcinomatosis, mainly because the lesions in the lung diminished. I am in favour of fungous disease.

DR. J. VOSS: I am intrigued by the mode of onset. I agree it is a polysystemic disease, but I doubt whether it fits *polyarteritis nodosa*. Polyarthritis in a female, associated with albuminuria, makes one think of disseminated *lupus erythematosus*. This could account for carditis, pneumonitis and arthritis, as fibrinoid necrosis could occur in almost any tissue of the body. There are one or two worrying features. One is that there is no apparent effusion. The second is that I am not sure whether the findings in the cerebro-spinal fluid are compatible with the disease. Perhaps Dr. Noad could help us here. The fibrinoid degeneration of diffuse *lupus erythematosus* is at one end of the scale of collagen "crack-up"; and, I suppose, if polyarteritis can cause cerebro-spinal fluid changes like this, so can lupus. A female of thirty-two presenting with polyarthritis, albuminuria and symptoms of involvement of all those other systems suggests to me the diagnosis of disseminated *lupus erythematosus*.

DR. NOAD: My experience of meningo-encephalitis or, shall we say, polioencephalitis, such as this woman had, shows

that it is very rare in disseminated lupus. Of course we are talking about much the same thing: in one, collagen is mostly affected; and in the other, the vessels are principally affected. In *periaratritis nodosa* the pleocytosis in the cerebro-spinal fluid is usually polymorphonuclear, more so than lymphocytic, and the protein level may be quite high, presumably because a lot leaks out from the surface vessels which are diseased. I have seen quite a few cases of diffuse *lupus erythematosus*, but I do not remember having seen one with marked cerebral symptoms, though I can imagine they could well be explained by vascular lesions in *periarteritis nodosa*.

DR. REIMER: Dr. Marsh, we felt you had more to say.

DR. MARSH: Not a great deal. We have the radiological appearances of miliary lesions throughout the lungs, an enlarged heart and no evidence of mediastinal or hilar glands, and I agree with Dr. Noad that this fits well a vascular disorder. I do not think it is fungous disease, else why should the heart be enlarged? I do not think it fits tuberculosis: nearly always there are enlarged glands by this time. I think the X-ray findings, as far as they go, support Dr. Noad's diagnosis.

DR. D. G. HAMILTON: I should like to comment on the cerebro-spinal fluid findings. I think we must remember that this woman had been having streptomycin for several weeks before the fluid was examined, and I think the findings are eminently compatible with tuberculosis which is partly controlled by therapy. I am rather surprised at some of those findings in the cerebro-spinal fluid of other cases of tuberculous meningitis as listed on the board. The sugar content seems high. We regard a much more marked fall than is recorded there as being a most valuable diagnostic feature of tuberculous meningitis in children. Chlorides we pay less attention to, as they do not fall very much until the disease is well advanced. The protein level is not necessarily very high in the early stages of the disease or when the disease has been partly controlled. Figures above 200 milligrammes per 100 millilitres we think suggest some degree of spinal block, and with figures as high as 800 and 2000 milligrammes we should be very suspicious indeed of blockage of cerebro-spinal fluid.

I was interested, too, in the arthralgia, and once again on the subject of tuberculosis, it is not uncommon to find arthritic pains in children with miliary tuberculosis or in tuberculous meningitis. Whether this happens in adults, I just do not know.

DR. REIMER: Dr. Hirst will now give us the pathological findings.

Autopsy Report.

A description of the post-mortem findings, illustrated by slides, was given by DR. E. HIRST. The following is a summary:

The patient had been an extremely thin (body weight 59 pounds), pale, young woman. There was only slight ulnar deviation of the fingers. No spindle-shaped deformity was seen. The abnormal findings were as follows:

Both pleural cavities were almost entirely obliterated by thin fibrous adhesions.

There was miliary tuberculosis of both lungs, both kidneys and the spleen, and bilateral caseous tuberculosis of the uterine tubes.

Beneath the craniotomy flap there was an extradural blood clot 7·0 centimetres in diameter and 1·7 centimetres thick. The convolutions were flattened. The ventricles were dilated and contained lightly blood-stained cerebro-spinal fluid. A few tubercles were scattered along the course of the basilar artery. The *pia mater* over the basal cisterns and the pons were thickened and œdematous. The interpeduncular fossa was filled with caseous material. Small punctate hæmorrhages were present in the frontal cortex and in the pons.

An incidental finding was an epicardial fibrous nodule in the posterior wall of the right ventricle.

Microscopically the presence of miliary tuberculosis was confirmed. The caseous mass in the interpeduncular fossa was a small meningeal tuberculoma.

Pathological Discussion.

DR. REIMER: Would you care to comment, Dr. Noad?

DR. NOAD: I can scarcely speak! As Hippocrates said, judgement is difficult and experience fallacious. I should like Dr. Hamilton to explain the negative first-strength Mantoux response—perhaps it was due to an anergic phase; the "fleeting suggestion" of neck stiffness; the poor response to streptomycin despite large doses—all of which, to my mind, point against a diagnosis of tuberculosis. Then there was the preceding history of arthropathy. Perhaps he would talk about these things.

DR. HAMILTON: I think the Mantoux reaction is very difficult to explain. Occasionally in children with miliary or meningeal tuberculosis we do get a negative Mantoux reaction, but rarely so. We regard it as a most important diagnostic test in tuberculosis of any sort. Presumably this woman had had tuberculosis for quite some time, and yet the reaction was weak.

PROFESSOR F. R. MAGAREY: I think Dr. Hirst said the woman had bilateral tuberculous salpingitis. That would have been there a long time and would have given rise to the miliary spread. This makes it even more surprising that the Mantoux reaction was negative.

DR. REIMER: Dr. Hamilton was asked to come along today to contribute to our discussion. Perhaps you would like to comment on some of the other features.

DR. HAMILTON: I do not regard neck stiffness as a necessary concomitant to the diagnosis of tuberculous meningitis. Once again, I speak of reasonably early meningitis. I remember coming into the ward one morning in England to find a child had been admitted the night before, and the registrar, a well-trained Irishman, had been concerned because the child had been cranky and irritable for a few days. He had therefore performed a lumbar puncture to exclude tuberculous meningitis in a child who, I thought, was only teething; that is the extent to which they are conscious of this disease in England. In this early stage of the disease it is common for there to be no neck stiffness at all. Later, of course, it is unusually present, but rarely is it as marked as in purulent meningitis or in most cases of poliomyelitis. Personality changes, scattered palsies, squint, but particularly irritability in a child who is vaguely unwell, these are the common early signs. Drowsiness soon follows, but if the child becomes comatose before the diagnosis is made, there is not much hope.

As for therapy, there is an enormous variation in opinion in different parts of the world. Basically treatment depends on the use of the three great antituberculosis drugs, streptomycin, para-aminosalicylate (PAS) and isoniazid. All schools would give at least two of those, except an occasional physician, including Professor Gaisford, of Manchester, who has treated the disease with nothing but isoniazid by mouth. And he got some good results. This was some time ago; perhaps his views have changed. Generally, in England, the three drugs are used together: injections of streptomycin for at least six months, intrathecal injections of streptomycin for four to six weeks, depending on how rapidly improvement occurs, isoniazid for a year or longer, and PAS possibly for eighteen months or longer. The Oxford workers have been giving all of these and P.P.D. as well, though this has been mostly given up where it has been tried elsewhere.

In America, on the other hand, the trend is to give up intrathecal streptomycin therapy but to use the other three drugs. The Americans rarely give intrathecal therapy of any sort. Who is right and who is wrong cannot be decided yet. There is no doubt that children are very much more comfortable without having a daily lumbar puncture. All get some good results, but all agree that early diagnosis is the most important factor for successful treatment. Lorber, of Sheffield, one of the best authorities on the disease in England, is very emphatic that if drug therapy is given at all, then all three drugs must be given. If only one hurdle is placed in the way of the bacillus, some will overcome it and be resistant; if there is a second hurdle, fewer will survive; and if a third hurdle is used, even if some bacilli become resistant to all three drugs, there will be very few live organisms left. This is a strong argument.

If one drug is to be left out, then I think there is no question that it should be PAS. The Veterans Administration in America published in 1954 what is probably the biggest series yet of cases of tuberculous meningitis, 287 in all. They were grouped according to treatment, and the groups whose treatment included isoniazid did so much better than those without it that there seems no doubt that it is an essential drug.

There is one other advance which, I think, is a very important and interesting one. That is the use of cortisone. We are taught, and quite rightly so, that we should make quite sure that the patient has not got tuberculosis before giving cortisone for any reason at all. We know that it sometimes causes the disease to flare up. Why, then, is it sometimes given in tuberculous meningitis? It is not given as a routine, but only to patients who are very ill and are not responding well, patients in whom we suspect that intracranial adhesions are forming which might lead to nasty sequelæ. Cortisone is given here in the hope of

reducing the formation of the scar tissue, to prevent loculation and obstruction of cerebro-spinal fluid. I think it probably has some effect, though no definite conclusion can be reached from the literature on the subject. In the few patients in whom I have seen it given I think it had some value. We give 300 milligrammes the first day, 200 milligrammes daily for a couple of days, 100 milligrammes daily for a week or so, and then gradually reduce the dose to finish the course in three or four weeks or longer.

Our experience with P.P.D., streptokinase and streptodornase in cases in which it was suspected that block was developing has been most disappointing.

In summary, then, I would treat tuberculous meningitis with intrathecal and intramuscular injections of streptomycin, with isoniazid and PAS. I would give intrathecal therapy for four to six weeks, intramuscular injections of streptomycin for at least six months, isoniazid and PAS for twelve months or even longer. If progress became disappointing, I would try the addition of cortisone.

DR. NOAD: I should like to add two comments. I would agree with Dr. Hamilton that in a child neck stiffness is often not as marked a sign as in the adult. In the adult, by the time the patient comes to hospital, neck rigidity is usually marked. Secondly, there remains unexplained, to my mind, why this patient had recurring bouts of pyrexia over the previous few months. Perhaps this was due to tuberculous salpingitis. The cause of these febrile episodes is one point we have not cleared up.

Diagnosis.

Acute disseminated miliary tuberculosis and tuberculous lepto-meningitis with internal hydrocephalus.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Repatriation General Hospital, Heidelberg West, on July 6, 1955. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staffs of the hospital. Part of this report appeared in the issue of May 26, 1956.

Cystic Parotid Tumour.

DR. N. H. HURSE showed a man, aged fifty-three years, who had been first examined in the surgical department in August, 1954. He had then had a cystic swelling in the left pre-auricular area about four centimetres in diameter. The swelling had been first noticed ten months previously, but had increased in size, mostly in the preceding month. A skin cancer on the left side of the forehead had been irradiated three and a half years previously. In September, 1954, the swelling was incised and was found to contain brown fluid and white necrotic-looking material. A small piece of the wall was removed for biopsy examination, and the following report on the tissue was supplied by Dr. G. F. Davies:

> The tissue is mainly atrophic parotid glandular tissue. Atrophic acini and ducts are placed in œdematous looking connective tissue in which there is some lymphoid infiltration. The ducts are not dilated. There is also some stratified squamous epithelium which forms an incomplete covering down one side of the tissue and has apparently formed the lining of the cyst described. In several parts the nuclei of this epithelium are hyperchromatic and irregular in size but no definite invasion can be seen and the appearances are not very suggestive of malignancy. In some parts there are degenerative changes with intracellular œdema. As far as can be seen from the examination of this section the lesion is a parotid cyst but the nature of the necrotic material is not explained. In view of this and the epithelial hyperplasia it is suggested that the patient be re-examined from time to time.

After operation a sinus persisted discharging clear yellow fluid and keratin-like material. The patient was given small doses of irradiation and, while receiving this, developed a left facial paresis. More intensive irradiation was commenced, and aspirated material was sent for section and examination. The following report was made on this material:

> The material aspirated from the parotid gland is mainly keratin, yet many nuclei still remain and these show considerable hyperchromatism and irregularity in size and shape. Several small clusters of cells show similar changes. In spite of the fact that the actual behaviour of these cells can not be observed their appearance is very strongly suggestive of squamous celled carcinoma.

The patient was now considered to have had a cystic degeneration of epitheliomatous pre-auricular lymph nodes secondary to the skin cancer. Deep X-ray therapy was given to the parotid from November 4 to 29, 1954, and the facial expression subsequently improved. In February, 1955, radon needles were implanted into the left parotid area. In June, 1955, a sinus had redeveloped in front of the ear, and on one occasion it bled freely.

Dr. Davies discussed other cystic parotid conditions including duct cysts, branchial cysts, cold abscess, adenolymphoma, cystic mixed parotid tumours, and degeneration in primary parotid malignant conditions. He also demonstrated relevant sections.

Recurrent Parotid Tumour.

Dr. Hurse then showed a patient who had presented in February, 1955, with five or six subcutaneous nodules spread along a scar over the left parotid gland. The nodules were half to one centimetre in diameter. In 1937 a parotid tumour about half an inch in diameter had been removed from behind the angle of the left jaw. It recurred three years later as two discrete nodules, which were removed in 1949. Six months later it again recurred as multiple nodules, one of which was removed in December, 1954, for biopsy, the following report being made by Dr. A. Brenan:

> The irregular cyst is partly filled with papillary tumour. This consists of a network in which there are many small tumour cells. Some portions are degenerate. There are one or two masses with a similar structure in the firm fibrosis stroma. One separate piece which apparently came out of the cyst is markedly cellular. The cells are mostly small round and spindle tumour cells suggestive of a small cell carcinoma.

In April, 1955, a course of X-ray therapy was given to the left parotid region, 3000*r* being given to the skin in three weeks. The nodules then decreased to half size and were excised in June, 1955, by Dr. I. Heinz. It was intended to give further irradiation after operation.

Naso-Pharyngeal Adenocystic Carcinoma.

Dr. Hurse's third patient, a man, aged seventy-three years, had presented in December, 1953, complaining of nasal obstruction present for nine months, as well as some difficulty in swallowing, and on several occasions a blood-stained discharge from the back of the throat. Examination of the patient showed a proliferative tumour arising from the left lateral naso-pharyngeal wall, with a large polypus emerging through the left posterior choana. Material punched from the naso-pharyngeal tumour was submitted to Dr. Davies, who gave the following report:

> Sections show the presence of some large masses of epithelial cells embedded in a delicate fibrous stroma. The cells are small, irregular and dark-staining, and have a cribriform arrangement, the interstices being filled with material resembling mucus. Most of the cell masses are well defined but in some parts narrow strands of cells have become separated and are apparently infiltrating the tissues. Tumours of this kind are often referred to as an adenocystic carcinoma. The presence of probable mucus suggests that it comes from glandular epithelium. Careful search reveals the presence of occasional groups of cells arranged round a minute lumen. Nasopharyngeal adenocystic carcinoma. An origin from mucus glands is suggested.

From December 26, 1953, to January 21, 1954, deep X-ray therapy was given to the naso-pharynx, with reduction of the tumour to one-half of its original size. In May, 1955, the nasal obstruction recurred with alteration in voice production, and a second course of X-ray therapy was given.

Dr. Hurse commented that the type of tumour concerned tended to persist or to recur locally over a long period of years, eventually involving lymph nodes and giving rise to visceral deposits.

DR. G. F. DAVIES demonstrated biopsy material from the patient and discussed other types of naso-pharyngeal tumour.

Demonstration from Eye Department.

A series of patients from the eye department were presented by DR. E. D. E. O'BRIEN, DR. W. C. F. O'HAIR and DR. E. G. GLASSFORD.

Two patients had diabetic retinopathy. The first had advanced disease with extensive retinal and vitreous hæmorrhage and loss of useful vision in both eyes. The second had early changes with capillary aneurysms and hard exudates in the macula region but no visual loss.

Another patient had an unusually large area of medullated nerve fibres extending to the temporal side of the macula and simulating exudative retinopathy of the circinate type.

Of two patients with optic atrophy, one had total unilateral primary atrophy, the sole sequel of meningococcal (cerebrospinal) meningitis. The other had partial optic atrophy following bilateral papilœdema associated with parietal astrocytoma. Two patients had bilateral tuberculous choroidoretinitis, both with extensive involvement at the posterior poles. One patient had responded well to intensive cortisone therapy with almost complete restoration of vision in both eyes. The second had responded poorly to the same treatment with partial recovery of vision in one eye only.

Three patients had had extensive bilateral corneal opacities. The first showed the results of corneal grafting. The second patient had mustard gas keratitis and was not suitable for grafting. The third had parenchymatous keratitis, which was suitable for grafting.

Other patients were presented suffering from the following conditions: bilateral pseudo-papilloedema due to colloid bodies in the optic papillæ, associated with angioid streaks in the retina; chronic myeloid leuchæmia with the typical fundus appearance; subacute secondary glaucoma with a deep glaucomatous cup and a very rapid response to "Diamox" therapy; Reiter's disease with chronic iritis and *iris bombé*; recurrent acute hæmorrhagic iritis with massive exudate and hæmorrhage in the anterior chamber.

Cutaneous Malignant Disease.

DR. W. W. LEMPRIERE showed a series of patients with various forms of cutaneous malignant disease—namely, basal-celled carcinoma, squamous epithelioma (cutaneous horn) and Bowen's disease (superficial epithelioma). The treatment of the various types of lesion was discussed.

Skin Diseases in Ex-Servicemen.

DR. F. BAUER showed a series of patients suffering from skin diseases which practically only occur in ex-servicemen.

The first group showed the aftermath of tropical lichenoid dermatitis of varying severity. The condition had been caused by taking mepacrine for control of malaria. The first patient had leucoplakia of the tongue and atrophic changes leading to fissuring of the palms and soles. The second patient suffered mainly from inability to sweat in the lower two-thirds of his body. The third patient suffered from patchy alopecia, loss of body hair, depigmentation of the skin, severe atrophic changes of the skin, smooth atrophic changes of the tongue, leucoplakia of the cheeks and inability to sweat (tropical anidrotic asthenia). He was unable to exert himself at all during the hot weather. Dr. Bauer commented that that was the most extreme degree of damage known, affecting all layers and adhera of the skin.

The second group had a creeping eruption caused by infestation with *Strongyloides stercoralis*. The eruption occurred in prisoners of war from Malaya, who suffered from an intestinal infection of Strongyloides and autoinfection of the skin by the filariform larvæ. Dr. Bauer discussed the differential diagnosis and pointed out that eruption was often misdiagnosed as urticaria. He showed "Kodachrome" pictures of patients with the above conditions and slides of *Strongyloides stercoralis* larvæ.

Spontaneous Expectoration of Caseous Tuberculous Lesions.

DR. W. LEIGH FORSYTH showed chest X-ray films of a series of patients in whom caseous tuberculous lesions appeared to have been expectorated spontaneously. Three lesions had disappeared between regular out-patient reviews without signs or symptoms of disease arising. No radiological evidence of spread of the disease had followed their evacuation, and no patient had received antibacterial drug therapy. Dr. Forsyth said that in each case it was obvious that the lesions were not recent. They had been present, unchanged, in X-ray films for many months at least before their disappearance. It was suggested that softening and evacuation of such lesions could occur without serious risk of spread of tuberculous disease in the lung.

Lung Abscess.

DR. W. A. MCKAY presented the clinical history, bacteriological findings and treatment and demonstrated serial chest X-ray films of patients with the following conditions: lung abscess due to *Staphylococcus aureus* treated with "Aureomycin" and streptomycin; lung abscess due to Friedländer's bacillus treated with streptomycin; lung abscess due to pulmonary amœbiasis treated with emetine; non-specific lung abscess treated with penicillin.

In addition he presented details of the following two examples of lung abscess associated with bronchial carcinoma: lung abscess due to bronchial occlusion by squamous cell carcinoma (with autopsy specimen); lung abscess in a "breaking down" peripheral squamous cell carcinoma (with a specimen obtained at lobectomy performed by Dr. J. I. Hayward).

Identification of Intestinal Parasites.

DR. A. R. PARKIN demonstrated methods used at the Caulfield Annex of the out-patient clinic in the identification of intestinal parasites (protozoa and helminths). Carefully chosen portions of fresh fæces were examined in saline, vitally stained with neutral red and stained in an iodine solution to demonstrate the presence of amœbæ in 33% of all patients. Definite identification of the amœbæ and cysts was made only after search of a preparation stained with iron hæmatoxylin. Examples of the stained organisms were shown. The results obtained in the years 1948 to 1954 were analysed, and comparisons from those years were exhibited showing the following striking features: (i) The fall in incidence of hookworm in that period was from 13% to 1·3%; the figures for other worms—strongyloides and trichuris—were similar. (ii) On the other hand the incidence of amœbæ remained virtually unchanged, *Entamœba histolytica* being found in 6% and 6·4% of patients in 1948 and 1954 respectively. The analysis also showed the value of four stool tests for *Entamœba histolytica*, for while only 40% of "positive" cases were diagnosed at the first test, 96% were confirmed by the fourth test and very few thereafter. Motion pictures also were shown of living entamœbæ, hookworms and threadworms. The films had been prepared in the department by Dr. Parkin.

Pathology Demonstration.

DR. R. B. MAYNARD and the staff of the pathology department presented a display with the following features: (i) A display of specimens showing invasion of the pulmonary artery by primary lung carcinoma. (ii) A large pituitary adenoma unsuspected clinically producing fairly gross hydrocephalus; the histological appearance was of the chromophobe cell type. (iii) Specimens (which were discussed) from a case of diabetes and Addison's disease. (iv) A demonstration of methods for differential staining of the pituitary gland. (v) A demonstration of biochemical methods of recent development, including (*a*) the use of quinine resin in a qualitative method for the detection of achlorhydria in patients unable to withstand intubation, (*b*) the estimation of serum copper content, (*c*) the estimation of serum magnesium content, (*d*) a rapid colorimetric method for the estimation of urea, (*e*) an easily manufactured syphon for pipette washing, and (*f*) electrometric titration. (vi) Bacteriological methods in the investigation of *Mycobacterium tuberculosis*: (*a*) pyrazinamide sensitivities, (*b*) a rapid method for the detection of avirulent strains of mycobacteria, (*c*) a laryngeal swab method for the detection of *Mycobacterium tuberculosis*. (vii) A demonstration of flame photometry, in which internal lithium standards were used.

Radiological Demonstration.

DR. A. J. T. ARAM and DR. W. R. C. BENNETT demonstrated methods of radiological investigation of the chest and gastro-intestinal tract. The value was shown of lateral sectional radiography in chest investigations in which other methods, including antero-posterior planigrams, had failed to provide the diagnosis. Thick section (three and six centimetres) planigrams as "scout" films in sectional radiography were shown, and it was pointed out that the short exposure time used for those planigrams might prove useful in demonstrating abnormalities in the region of the heart and great vessels as heart movement was minimized.

In the gastro-intestinal demonstration, the main emphasis was on cases in which there was kinking and rotation in the gastro-intestinal tract. In particular a series of cases of partial volvulus of the stomach was shown, the different types of rotation being well demonstrated. The high position of the transverse colon in relation to the stomach in such cases was well seen. A case of sigmoid volvulus diagnosed pre-operatively on the plain film of the abdomen was included in the "kinking and rotation" series. Amongst other gastro-intestinal conditions included was a typical example of gastric mucosal prolapse through the pyloric canal with a filling defect in the base of the duodenal cap.

Films on Anæsthetics.

DR. J. EGERTON WILLIAMS, from the anæsthetic department, showed two films. The first, which was on "'Pentothal' and its Application", had been prepared by the staff of the Wesley Memorial Hospital, Chicago, Illinois, and was sponsored by Abbott Laboratories Proprietary, Limited. The film ran for some thirty minutes and showed in colour a good description of the chemistry and pharmacology of "Pentothal" and its practical application to different patients suffering from different conditions and in different states of health, finishing with a complete record of its use in the operating theatre.

The other film, which ran for some ten minutes, was on the use of relaxants, particularly succinyl choline chloride ("Scoline") in conjunction with "Pentothal" for shock therapy. The film had been photographed by the anæsthetic department in the psychiatric wards of the hospital.

Leucotomy.

DR. J. M. HURT and DR. I. ATKINSON presented a demonstration on 10 recently performed limited leucotomies (orbital undercutting). It was pointed out that since Christmas, 1954, nine limited leucotomies had been performed. The tenth had been on a paranoid schizophrenic patient in April, 1954. The indication for operation was mainly tension, as seen in anxiety states, obsessive neurosis and paranoid schizophrenia. One patient had involutional melancholia which could not be controlled by electroconvulsive therapy and drugs. Most patients were incapacitated with poor work records and often attempts at suicide. The patients were carefully selected, being examined by two psychiatrists, a neurosurgeon, a neurologist and a psychologist; in addition, routine tests were carried out, such as the Wassermann test, X-ray examination of the skull and electroencephalography, and a social history was obtained. Post-operative clinical findings included freedom from the incontinence and confusion so common with the classical operation. Immediately after the operation the patient complained of the pressure of the bandages; and when those were removed after one week, the patient often became flippant. That phase lasted a week or so; then the patient showed lack of drive, easily became fatigued and was impatient at being in hospital—for example, complaining that occupational therapy was a waste of time and wishing to get back to work. So far the patients expected to return to work had done so without difficulty.

The psychodynamics of the patients appeared to be unchanged, but they were no longer overwhelmed by emotional outbursts. Emotional ties with their families were often off-handed, and that upset their wives, who despite explanation expected their husbands to be perfect. Both the schizophrenics had denied the fact that the operation had taken place.

DR. C. JORGENSEN and MR. R. LEONARD had carried out an extensive psychometric follow-up examination and had found no significant loss. Forethought and planning appeared unchanged.

The concluding comment was made that the limited operation appeared to gain all that the classical operation did without the drastic losses and alteration in personality. Only two rather hysterical patients had declared that they were no better at the date of the demonstration. Since then one of them had improved a great deal. No undesirable personality changes had occurred. Dr. Hurt and Dr. Atkinson said that they would prefer to wait at least one year after the operation before definitely assessing the patients, as the literature indicated that they were "plastic" for that period.

Resuscitation.

DR. L. E. BULEY presented a demonstration from the resuscitation department.

Statistics were displayed relating to the activities of the blood bank and resuscitation unit over the past five and a half years. The following figures were quoted for the calendar year 1949, the calendar year 1954 and the six months from January to June (inclusive), 1955, respectively: number of donors bled, 2267, 2656, 1301; number of transfusions of blood and derivatives given, 618, 847, 374; number of pints transfused, 1356, 1841, 1007; number of electrolyte solution infusions given, 457, 756, 430; number of litres infused, 1670, 2703, 1755. It was pointed out that the blood bank at the Repatriation General Hospital, Heidelberg, was unique in the fact that it was entirely autonomous. It obtained its blood chiefly from organized donor panels from branches of the Returned Sailors, Soldiers and Airmen's Imperial League of Australia, from sporting bodies and from repatriation staff and serving service personnel. It contributed to the pool which supplied the Commonwealth Serum Laboratories with blood for processing as serum, serum albumin *et cetera*.

The composition and milliequivalence tables of stock fluids for intravenous use were presented, and the production was shown of fluids of desired milliequivalents and osmolarity by graduated addition of supplements to stock solutions.

A project was presented embracing the scope of resuscitation therapy as interpreted at the hospital. The field was covered under sections dealing with "Assessment", "Correction or Prophylaxis of Physiological Depletions and Excesses", "Administration of the Materia Medica of Resuscitation" and "Miscellaneous Special Measures". Each section was illustrated by a number of examples based on actual cases—for example, "The Management of Anuria with Hyperkalæmia and Uræmia". The "Special Measures" section dealt with such diverse activities as the management of barbiturate poisoning, the investigation of transfusion reactions, plombage of œsophageal varices and prophylaxis of post-operative pulmonary complications (with special reference to partial gastrectomy) by the use of bronchial detergents.

Coupled with all sections was a display of transfusion and resuscitation equipment, including equipment being developed at the hospital, such as a remote-reading drip-rate indicator, and of the new items of the *materia medica*, such as kationic exchange resins.

MR. T. TILSEN, biochemist, demonstrated the rapid determination of serum electrolyte levels with the Beckman flame photometer, using lithium standards and the manometric Van Slyke apparatus. Dr. Buley explained the practical application of the results to therapy.

Medical Societies.

PÆDIATRIC SOCIETY OF VICTORIA.

A MEETING of the Pædiatric Society of Victoria was held at the Royal Children's Hospital, Melbourne, on March 14, 1956.

Social Study of Accidental Poisoning.

MISS JEAN ALLAN, Dip.Soc.Stud., V.I.H.A., S.R.N., of the Department of Clinical Research, presented the results of a social study of accidental poisoning (see M. J. AUSTRALIA, February 11, 1956, page 213).

Miss Allan said that from the patients studied it seemed that poisoning was a relatively common accident in young children, especially between the ages of one and three years. Approximately one child in every 130 children between the ages of one and three years took a poisonous substance. Such accidents were very distressing to parents, who frequently felt that they had been careless or neglectful. Negligence or carelessness on the part of the parents was an uncommon cause of poisoning. Of the mothers 79% were good and careful and handled their children well. Of the children 63% came from good standard homes, and of the fathers 79% were professional or business men or skilled tradesmen. The smaller number came from the poorer families who belonged to the labouring or semi-skilled groups and who had a lower material standard of living. Three important factors relevant to prevention emerged from this social study: first, the larger number of children who took poison were active, curious children; second, many of the substances ingested were readily accessible to the children; third, it was not realized by parents that a number of common household substances were dangerous.

Miss Allan said that prevention of a considerable number of accidental poisonings would probably best be achieved by education of the parents on the frequency and danger of poisoning for the child of one to three years of age, especially for the inquisitive child, and by provision of suitable storage for and suitable labelling on all poisonous substances. Education would probably be best carried out personally, preferably by a visiting nurse and family doctor.

DR. HOWARD WILLIAMS, in opening the discussion, stressed some of the outstanding features of the paper. He said that it appeared that those interested in child welfare must do something about the question of poisoning, and the emphasis of the work should be on prevention. Workers for child health had met the problem of infection and nutrition, but the emphasis should now be shifted to understanding the child from a wider point of view—in other words, understanding the child as a social human being.

DR. D. SHIELS asked Miss Allan why she thought that broadcasting would not be a useful means of distributing propaganda against poisonous substances. He went on to say that he thought surprising the faith people had in governments. The attitude was: "It must be all right; it is sold openly." He said that many extremely poisonous substances were not so labelled at all and cited a furniture polish, and substances containing aniline dyes. He thought that kerosene should be labelled as a poison. He suggested that the Pædiatric Society of Victoria should make representation to the Pharmacy Board or the Health Department to control the question of correct labelling. However, new legislation on the matter would be difficult to obtain, as it might mean altering the *Health Act*.

He thought that one of the reasons why cases of poisoning might come from better homes was that such places contained better supplies of all the relevant new products. Modern improvements were adding to the hazards of life. He stated that there had been 17 cases of lead poisoning in the past three years, including three deaths.

DR. BYRON STANTON said that they must recognize the fact that the present age was one of drug-taking. Aspirin, phenobarbitone and amphetamine were almost part of the national diet, and therefore the adventurous child would by sheer imitation do the same thing as adults—for example, take things out of bottles.

He agreed with Dr. Shiels that compounds found around the house were becoming more poisonous. The National Health Scheme also added to this, as drugs were prescribed in excess of what was needed. An additional hazard was the fact that the nature of the drugs prescribed was never written on the bottle. It was not considered ethical to label them "poison" or to say what they were. He thought that patients should be told what the medicine was. It should be labelled as "poison" and "to be kept away from children". Medical practitioners should move in the matter, but chemists might resist the move.

Each State had its own *Poisons Act*, but there was a move to establish a uniform *Poisons Act*. It was proposed to place kerosene on the poisons list, and discussion was in progress as to what colour it should be made; possibly it would be black.

More modern poisons were dangerous, and many were without specific antibodies. It was proposed to have one pharmacy in every municipality which would have a well-stocked poisons emergency cupboard. This would include a list of poisons and antidotes, stomach pumps, resuscitative measures and antidotes. Every public hospital should have the same thing, and it should be constantly reviewed.

DR. BARBARA MEREDITH said that she knew that more emphasis should be placed on the prevention of poisoning in the work of infant and child welfare centres. Recently infant welfare sisters had been given education regarding accidents, and in schools lectures were given to the children, but perhaps insufficient emphasis had been given to poisons. She thought that a leaflet should be written and distributed through the infant welfare centres. Also, perhaps one health week could be devoted to accidents and poisonings. She said that all mothers were having an increasing burden placed on them and wondered whether Miss Allan's study showed that the accident happened during any stress to the mother.

DR. KATE CAMPBELL stressed the necessity for a campaign of prevention. She thought that the welfare centre was the natural centre for this, but a visual demonstration there, with a list of common poisons, would probably be better than a leaflet.

DR. V. COLLINS thought that all forms of propaganda should be used, including the radio, posters, demonstrations and dissemination of knowledge by doctors and nurses who might see the difficulties in the home.

DR. W. RICKARDS, in commenting on Miss Allan's use of patients with infantile diarrhœa as a control group, said that when Flanders Dunbar in her study of psychosomatic disease had chosen accident patients as a control group, they were found to be rather a specialized group. The concept of accident-proneness developed from that. He was interested to see that Miss Allan had thought that the children had some personality and behavioural features in common. He asked whether that observation was based on a visit to the mother after the accident, or whether she had followed the mother and child later.

Miss Allan, in replying to the questions, said that she thought that radio propaganda might be useful, but not as the particular way of education. Many mothers of one to three year olds had little time to listen to the radio. Propaganda of that sort would have to be constantly reiterated over the years. Education should be incorporated into the general child guidance programme.

To Dr. Meredith, Miss Allan said that only three mothers gave poisonous substances to the child in place of medicine when they were under stress. She had not made a particular study of the personality patterns of the other mothers in this group of children.

To Dr. Rickards, Miss Allan said that her statements regarding the personality of the children were based on what the mother said about the child at the time of the interview, which was shortly after the child had recovered, as well as on her own observations of the child's behaviour during the interview in the home. She had not as yet followed the children any further.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

BEER.[1]

[From the *New South Wales Medical Gazette*, June, 1875.]

THE Germans have begun to take cognizance of the quantity of beer retailed and to compare it with the quantity which could have been made with the hops and malt on which duty has been paid. The result has somewhat astonished politicians and proves that the custom of employing substitutes is more general than has been believed.

The extremely deleterious quality of much of the beer sold in New South Wales and the abeyance of any authority to enforce the law as regards selling beer to persons already intoxicated by it, render it desirable that some attention should be devoted to the subject in this colony. We believe that finances of the country would be as much benefited as the health and morals of the people if adequate measures were adopted to prevent adulteration. The custom of granting a licence to every applicant regardless of local necessities is destructive of all honour in the trade. To avoid insolvency the smaller publicans employ female decoys and use cheap adulterations.

Special Correspondence.

LONDON LETTER.

BY OUR SPECIAL CORRESPONDENT.

West Indians in Great Britain.

THE continued influx of West Indians into Great Britain is causing problems for both countries. Migration from the West Indies is not a new phenomenon, but its direction has changed owing to the closing down of alternative areas such as the United States and Panama. At the present time there are about 40,000 West Indians living in Britain, and it is estimated that another 15,000 will arrive during the year. The Governments of the West Indian Islands, particularly Jamaica, are concerned at the loss of their most enterprising young men and women, who are needed for their own economic development, while the British local government authorities are worried by the exacerbation of the housing problem. The arrival of the immigrants in Great Britain creates no unemployment problem. The immigrant never fails to find work. There is a demand for West Indian labour in industry and the transport services, although most immigrants have to be content with subordinate jobs. Coloured men are seen with increasing frequency on the London buses and on the stations of the underground. Coloured women are quickly absorbed into the hospital services as orderlies or trainee nurses.

The difficulties which arise are mainly due to the lack of knowledge by Britons and West Indians of each other's social background and aims. Problems arise from the colour

[1] From the original in the Mitchell Library, Sydney.

difference, the difference in skill between the West Indian and the British worker, the change in climate, the housing shortage and the high cost of living. The future may also produce further difficulties—for example, if the present rate of immigration continues, a point will be reached when Great Britain may be unable to absorb any more. Should a trade recession occur, the West Indian is likely to be the first to lose his employment.

Last year the Government of Jamaica sent over two representatives to study conditions under which immigrants live and work in Great Britain. Their report recommends, *inter alia*, the establishment of an educational service in the West Indies and the setting up of a welfare service for West Indians in Great Britain.

The Health of Farm Workers.

The agricultural worker in Great Britain is no longer poorly rewarded for his labour, and the farming industry is more economically secure than it has ever been. Attention has now been directed to the health and welfare of persons employed in agriculture and forestry. The Government has recently introduced legislation to implement the recommendations of a report of a committee of inquiry made in 1949, providing for the safeguarding of workers from injuries from machines, from the handling of animals and from the risk of falls and sprains. It provides that a young person shall not lift a load so heavy as to be likely to cause injury and prescribes the maximum weights which may be lifted, carried or moved by workers. There is provision for the prohibition of children under thirteen years of age from riding or driving certain vehicles, machines or implements used in agriculture, and also for adequate sanitary arrangements and washing facilities.

Food Hygiene.

The increased incidence of food poisoning in Great Britain has been the subject of comment in the medical and lay Press and is now receiving the attention of Parliament. New powers under the *Food and Drugs Act* are being sought to strengthen the hands of the local authorities to secure high standards of hygiene in the food and catering industries. In 1954, 6016 incidents of food poisoning were recorded, an increase of 739 over the previous year. Infection by Salmonella organisms continues to be the chief cause of food poisoning, but in a high proportion of cases (39%) no specific cause could be ascertained. Thirty-four patients died, 27 from infection with *Salmonella typhi-murium*. Among the foods consumed, meat, particularly when processed or made up, was the chief vehicle of infection, and in half the cases investigated the food was consumed outside the home, in canteens at school or places of work. The new regulations require certain standards before a certificate is granted by the local health authority for the use of premises where food is prepared or consumed. The use of newspapers for wrapping food, other than uncooked vegetables, is prohibited. Certain foods which provide a good medium for bacterial growth—for example, pre-cooked foods—must not be kept at a temperature at which bacterial multiplication is greatest. The Catering Association of Great Britain has complained of the short notice given to the industry and the cost involved, and believes that the dangers of food poisoning in relation to commercial catering establishments have been greatly exaggerated.

The Work of the General Practitioner.

An interesting analysis of the clinical records of 10 general practitioners over a period of two years has been published by the Registrar-General as Number 9 of a series of "Studies on Medical and Population Subjects". This inquiry, of the nature of a pilot study, was designed to test the practicability of using the clinical records of general practitioners as a source of morbidity statistics. Information obtained from so few practitioners cannot be regarded as representative of all general practice, but in the absence of more comprehensive information, the results are still of value.

In all, 13 practitioners in 10 practices with a total patient list of 37,000 took part. In these practices the doctor was consulted by about two-thirds of the number on his list each year. Each patient came, on an average, for about four consultations each year (men 2·6, elderly men 5·0, elderly women 5·7). Respiratory diseases caused 24% of all consultations, and digestive and skin conditions 7·5% each; 5% of consultations were given for reasons other than sickness—for example, general medical examinations, routine maternity examinations, and social and domestic matters.

The experience gained in this inquiry has helped to pave the way for a joint survey by the College of General Practitioners and the General Register Office in which some 170 practitioners are now engaged voluntarily in keeping standard records for twelve months. These records will be available for statistical analysis. The new inquiry will cover nearly 1% of the population of England and Wales.

Mental Health.

A pictorial exhibition on mental health is now touring the country. Its objects are to clear up popular misconceptions and ignorance about mental illness and to recruit nurses for the under-staffed mental hospitals. A change has already taken place in the public attitude towards mental disease, and it is becoming more freely accepted as an illness capable of remedy and not a social disgrace. Of those who enter mental hospitals today, 71% are voluntary patients, compared with 45% ten years ago. Moreover, because people seek treatment at an earlier stage, treatment is more successful. Nearly three-quarters of all new patients are discharged within a year, and the average length of stay is now only 1·9 months. There is a growing desire to increase the opportunity for early treatment by allowing a person to enter a mental hospital without the formalities required by the *Lunacy and Mental Treatment Acts*—that is, without any more formality than is required to become a patient in a general hospital.

There is still a grave shortage of mental nurses. The shortage creates unsatisfactory working conditions for the existing staffs of the mental hospitals. A demonstration by nurses took place in Manchester recently at the Town Hall, where the exhibition was staged, to draw attention to the "appalling" pay and conditions in the mental hospitals. Complaints were made of the low rate of pay and long working hours, up to sixty hours per week, without overtime. The ratio of nurses to patients in mental hospitals is 1:8, compared with the 1:2 in general hospitals. The old idea of the nurse as a mere custodian is passing, and mental nursing today gives opportunities and responsibilities not inferior to those of a general hospital nurse.

Visiting Children in Hospital.

The number of hospitals in Great Britain which now allow daily visiting of child patients has trebled in the last few years. Only 10% now restrict visiting to one day a week or less, including 28 hospitals which still prohibit all visits save in emergencies. Many of the traditional difficulties have been overcome, and daily visiting is almost universally accepted. Some hospitals which do not allow this regard it as undesirable to admit visitors on days when there are operating sessions, but the Central Health Services Council has advised the Minister for Health that there is no general reason why all visiting on these days should be prohibited. The visiting of children admitted for tonsil operations may not be necessary when the child's stay is twenty-four hours; but it is undesirable for a child to be separated from its parents if it is in hospital for three or four days, and visiting should be permitted unless there are special dangers of infection.

One of the main problems concerns infectious diseases hospitals, where the essential aim is the protection of the public. Subject to certain safeguards frequent visiting can be allowed, and in a number of such hospitals it is already allowed. Some of the safeguards suggested are the presence of sufficient nursing staff to ensure close supervision of the conduct of visiting parents, the wearing of protective clothing by visitors, the application to them of the routine measures for the prevention of the spread of infection, and the limitation of visitors to parents or guardians. The ultimate decision on the question of permitting visitors must depend on individual circumstances in relation to each patient and must remain with the medical officer of the hospital.

Maternity Homes.

A situation involving the mother-child relationship occurs in maternity homes and hospitals. The Maternity and Midwifery Advisory Committee of the Ministry of Health recommends that babies should be placed in cots beside their mothers' beds and not together in a separate ward. This enables the mother to get to know her child, avoids the frequent changes of environment and temperature for the baby, and reduces the risk of infection. There is need, however, to study the mother's need for rest and quiet.

A trial of the new method was carried out at the Scarborough Hospital, and the matron, making her round the next morning, found the whole ward in a state of tension. The mothers had had a very disturbed night, and the new

babies were restless and crying. After this experience, cross children were taken to the nursery, and the maternity ward was more peaceful and the mothers were happier. In the matron's view, the experiment would be successful in single room wards, where there would be no distractions.

Correspondence.

"POLIO."

SIR: Today I was cast into the profoundest melancholy when I opened a circular, from a body which included doctors, about something they called "polio". "Immunise against polio", they advised, and no nonsense about full stops after "polio". Glancing very casually, swiftly and angrily into other enclosed matter, I gathered they were talking about poliomyelitis, derived from *πόλιος*, grey, and *μύελος*, marrow, the word then obviously meaning inflammation of the grey matter of the (spinal) cord (marrow). If people in their repulsive barbarity talk about "polio" as a disease, they must necessarily mean the "grey disease" if the first "o" is short, or a disease associated with selling if it is long (*πωλέω*, I sell). Now, I know of no "grey disease", and some selling is, of course, a disease with paralysis of the ethical system. But the selling disease has, I should say, no relevance to poliomyelitis. The first "o" in poliomyelitis is short, as in policy or politics (*πόλις*, a city), while in the Greek word "to sell" it is long, as in pole; and this is the quantity given to "polio" by the sad routiners who jockey the discs for the Australian Broadcasting Commission when in fact they are talking of poliomyelitis, in which the first "o" is short, as the Greek root shows.

Now, some people say poliomyelitis is a cumbersome word, but we have no better name for the disease, and what about "imperturbability", "ætiologically" and, let us say, that quasi-barbarism "dedifferentiation?" Are we to say "imperturb", "ætio", or "dediffer", instead of saying the words out in full and writing them properly?

I loathe barbarism in all forms, not least in language. I loathe slipshod work and ways, and I especially loathe the medical man who is imprecise in language; he is sure to be like that in his work.

Of course, this accursed "polio" is not quite, but nearly, as revolting as "T.B." for tuberculosis.

You are, Sir, our last citadel of precision and taste; do not abandon us to the barbarians any more. Of course, newspapers are made by and for luckless people in a hurry, but I cannot see why their debased verbal currency, home-made at that, like the current "fivers", should so much appeal to us that we adopt it. Our dignity and taste should be above such derelict habits of thought and speech.

Yours, etc.,

J. V. DUHIG.

Ballow Chambers,
Wickham Terrace,
Brisbane, B.17.
May 10, 1956.

AN UNUSUAL SIDE EFFECT OF "DIGOXIN".

SIR: Last year Dr. Clyde Davis (M. J. AUSTRALIA, July 30, 1955) drew attention to the occurrence in a patient of his of an unusual and rare side effect of "Digoxin", which was being used for a heart condition: dimness of vision was noticeable while the drug was being used.

Now, in my practice is a man, aged fifty-five years, who constantly requires digitalis in some form, and the only preparation he can take without severe nausea and illness is "Digoxin" in tablet form. For a long time he could take only one tablet (0·25 milligramme) daily, but he can manage two tablets daily as a general rule now. When he takes two tablets daily he complains that his eyes are affected: there is no dimness of vision, but there is a "flickering of vision at the edges", occurring at any time, but especially on coming from shade into bright light. Sometimes, too, there is loss of focusing ability, and cars in the street "appear blurred"; and there is occasional but transient "double vision" (diplopia).

On occasions in the mornings he notices a brownish spot with a lighted edge in front of his eyes (either eye or both), apparently scotomata; this lasts some seconds, but does not appear regularly.

The symptoms persist while he takes two tablets, but improve and disappear on one tablet daily. He appears to be extremely sensitive to other preparations of digitalis with which I have tried to treat him.

Martindale in the 1952 "Extra Pharmacopœia" (page 499) records occasional visual symptoms with digitalis overdose and that "Digoxin" can cause the same symptoms, though it is less toxic. In the list of visual symptoms, flickering and scotomata do not appear.

Shortridge, in this journal (August 20, 1955), said that visual symptoms with "Digoxin" are well known to ophthalmic surgeons. In a letter to Dr. Davis (M. J. AUSTRALIA, July 30, 1955), Messrs. Burroughs Wellcome and Company mention diplopia and scotomata, and say that, although "visual disturbances are not rare as a sign of digitalis poisoning", "to the best of our knowledge no such case has so far been reported from the use of Digoxin".

My patient is a man extremely susceptible to any form of drug therapy, as well as to digitalis and its preparations, and perhaps that is the reason for the well-marked symptoms in his case. I do not think that these symptoms are familiar to most doctors, and only after Davis's letter does it appear that any note has been taken of them.

Yours, etc.,

CAWLEY MADDEN.

517 Pacific Highway,
Killara,
New South Wales.
May 11, 1956.

THE TREATMENT OF ULCERATIVE COLITIS.

SIR: I would like to have the opportunity to comment again on Dr. Haynes's interesting views on this subject.

In his recent letter (M. J. AUSTRALIA, April 28, 1956) he elaborates his concept of the neurotic basis of ulcerative colitis and goes further to attribute the same cause to "at least thirty relatively common diseases of doubtful ætiology".

It would add to the interest of his comments if he would tell us how he arrived at this conclusion. Only the unduly credulous would accept this explanation as it stands.

In view of his remarks about "the evidence I have found in these cases" it must be presumed that his complex concept has a logical explanation. The great majority of students of ulcerative colitis are waiting or looking for an explanation of its cause. Dr. Haynes's explanation may be the one the world is waiting for, but until he produces his evidence surely the only healthy regard for his concept is one of scepticism.

Yours, etc.,

J. W. GRAHAM.

Sydney,
May 5, 1956.

Obituary.

ERROL SOLOMON MEYERS.

ERROL SOLOMON MEYERS was born on August 9, 1890. He died on February 11, 1956, having lived a happy life of courage, toil and achievement.

He began his education at the Southport State School, and after winning a scholarship attended the Brisbane Grammar School. Here the love of learning and eagerness for knowledge which he had early displayed were given full rein under the inspiring teaching of such great masters as the late Reginald Heber Roe and F. S. N. Bousfield. He was a keen student of science, and of the classics, taking both Latin and Greek, for which disciplines he had a genuine and lasting appreciation.

A further scholarship took him to the University of Sydney, enabling him to achieve his long-cherished ambition to become a doctor. He graduated in 1914, winning a prize for clinical medicine.

He enlisted in the first Australian Imperial Force and served as regimental medical officer to the Forty-first Infantry Battalion. His comrades in the field came to hold him in high regard. In the words of the history of the Forty-first Battalion: "The Regimental Medical Officer, Captain E. S. Meyers, earned the gratitude and admiration of all ranks, often dressing cases under heavy shell-fire."

He was twice gassed, and returned to active service as commanding officer of the Eleventh Field Ambulance with the rank of major, taking part in the final assault on the Hindenburg Line.

After pursuing post-graduate studies in England, he returned to Australia in 1919. He married, and entered practice in South Brisbane. In 1921 he was appointed honorary assistant surgeon to the Brisbane General Hospital, at which institution he had been a resident medical officer prior to his enlistment in the Australian Imperial Force.

Two guiding principles of his life were the welfare of his patients and the advancement of medical education. It was with these aims in view that he had early realized the need for the establishment of a medical school in Queensland, a need which had been similarly exercising the minds of several of his colleagues. To the achievement of this ideal he devoted intense enthusiasm and energy, but it was not until 1936 that the goal was secured.

In 1922 he became honorary demonstrator in anatomy to students at the Brisbane Dental Hospital, a function which he continued to carry out for over a quarter of a century. For his services to dentistry in Queensland in this and in other regards he was made an honorary life member of the Australian Dental Association. In this work he not only gained the satisfaction of aiding a sister profession, but also saw it as a step towards the ultimate aim of a medical faculty. In 1926, under the provisions of a new *Medical Act* of Parliament, he became the first licensed teacher of anatomy in Queensland.

The task of establishing a faculty of medicine proved to be a difficult one, but in it he had the wholehearted support of three other worthy men, Dr. J. V. Duhig, the late Professor E. J. Goddard and the late Dr. Ernest Sandford Jackson, each of whom had a decisive contribution to make.

In 1932, after some ten years of unremitting effort on his and his colleagues' part, Dr. Meyers, in his presidential address to the Queensland Branch of the British Medical Association, called for immediate action to establish a faculty of medicine. He outlined a practical plan for the achievement of this aim. However, several years were to elapse before the necessary governmental action could be taken, and in 1936 the Faculty of Medicine in the University of Queensland, the Medical School, came into being.

As an active part-time member of the teaching staff, Dr. Meyers lectured and demonstrated in anatomy and in clinical and operative surgery. In 1941 he was elected to the deanship of the faculty, a somewhat rare honour, inasmuch as the post was usually the prerogative of a professor and not of one of only lecturer status. He continued to be reelected to the position of Dean of the Faculty of Medicine for the next twelve years, itself a significant record, bearing an increasing burden of work through the war years, and in the post-war period of unprecedented growth of the school. His every endeavour was expended in guiding the destiny of the faculty which he had helped to found, and in safeguarding the best interests both of the students and of their future patients.

In 1946 the position of honorary professor of social and tropical medicine fell vacant, and in response to requests he undertook these duties also, assuming the status of professor from then onwards.

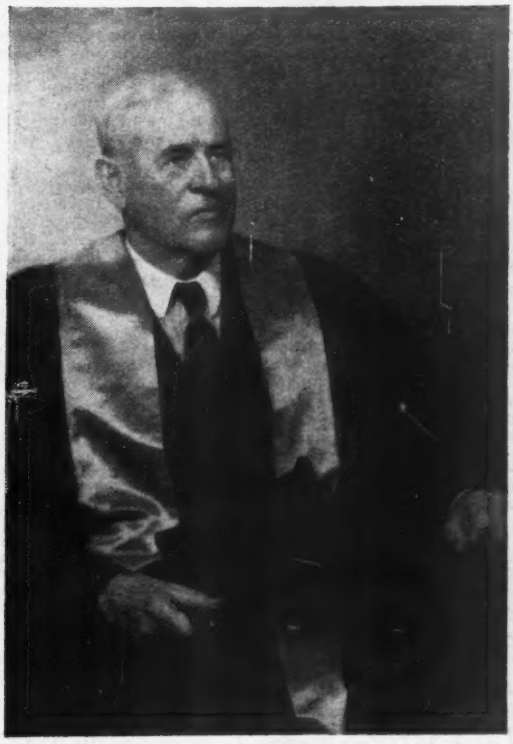
His prestige as a medical educator was considerable and increased with the passing of the years. In 1953 he was honoured to be one of the two Australians who were invited to be speakers at the Inaugural World Conference on Medical Education held in London.

He took a practical interest in the welfare of all his students, and apart from his lucid teaching, gave unstintingly advice and assistance to many grateful students. He maintained a friendly but unmistakable discipline in his classes, yet treating each student as his fellow man. He was held in the highest esteem by them, and was made an honorary life member of both the Dental and Medical Student Societies.

Although he spent many years in general practice, his leanings were towards general surgery. In 1926 he became senior honorary surgeon to the Brisbane General Hospital, which post he held until 1943, and was one of the early Fellows of the then newly formed Royal Australasian College of Surgeons.

He strove always for improvement. His activities in post-graduate education began in 1922, when he formed the Brisbane Hospital Clinical Society. His far-sightedness and altruism led to the expansion of this type of meeting, and the late Dr. S. F. McDonald wrote in 1946: "Twenty years ago, largely owing to the enthusiasm and determination of Mr. E. S. Meyers, the Medical Post-Graduate Committee came into being."

He was a gifted anatomist and a surgeon of outstanding ability. He contributed over the years to basic research in anatomy, and pioneered the surgical operations for relief of cancer of the neck in Queensland. His original contributions to medical literature were commended by authorities both interstate and overseas. He read widely and intelligently, and was quick to appreciate and utilize the best advances in diagnosis and treatment. In Brisbane he was the first to perform ureteral catheterization, and the first to perform the operations of phrenic crush and thoracoplasty.



Despite the pressure of work, he found time to take part in other matters of community interest. He played an active part as an officer of the Australian Army Medical Corps, receiving the Long Service Medal and the Volunteer Decoration, and retiring with the rank of lieutenant-colonel. For some years he was examiner in first aid for the Railway Ambulance Competitions. He was a foundation member of Legacy in Queensland, and here rendered much useful service. He was for three years consecutively honorary secretary of the Queensland Branch of the British Medical Association, and once its president, and served for a busy period on the Federal Council of the Association. In the early years of the use of radium in the treatment of cancer he was Curator of Radium in Brisbane, and a member of the Radium Committee of the Brisbane Hospital. He was a member of the committee of the early Federal Cancer Conference, whose aim was to coordinate and improve research into and treatment of cancer in the several States.

He pioneered social medicine in Queensland, the State whose university was one of the first in the Empire to establish a chair of social medicine. He sought constantly to bring to the notice of his fellow citizens ideas of proven value.

In the years of the second World War the burden of work increased considerably. Although he lost the companionship and help in this period of his three sons, who enlisted while medical students, two to serve overseas with the Royal Navy and Royal Australian Navy, and one as aircrew with the Royal Australian Air Force, he was sustained by the love and devotion of his wife and daughter. His health slowly

began to deteriorate. In 1946 he suffered a severe attack of cardiac asthma, the first of many more to follow. Supported as he had been so admirably throughout by his wife, he bore his illness bravely and uncomplainingly, his constant regret being his inability to do the many things which he knew were yet to be done.

To the end he displayed the courage and steadfastness which had characterized his life, and which had earned for him the respect of his comrades in France and Flanders. Honesty, industry, humility and compassion formed the background of his strong and fearless character, without which the ideals for which he strove could not have been achieved. In all truth and sincerity may it be claimed that he rendered outstanding services to medicine and to his fellow men.

As was said of another great surgeon: "He lived to enjoy the satisfaction not only of gazing retrospectively on his own brilliant pioneer achievements, but of looking forwards into the promising vista which his work had opened up. Well might he have claimed in the words of Valiant-for-Truth 'Yet now I do not repent me of all the trouble I have been at to arrive where I am. My sword I give to him that shall succeed me in my pilgrimage, and my courage and skill to him that can get it . . .'"

ISABELLA YOUNGER-ROSS.

WE are indebted to Dr. W. Barbara Meredith for the following appreciation of the late Dr. Isabella Younger-Ross.

Isabella Younger, a Victorian by birth, commenced her medical course in Melbourne, but later went overseas and graduated from the University of Glasgow. Her post-graduate training included a period as assistant to Dr. Eric Pritchard, a pioneer in baby health work. She returned to Melbourne in June, 1917, and immediately set to work to explore the possibilities of establishing "baby clinics" in Victoria. With Mrs. Hemphill and Mrs. V. Ramsay, leaders in philanthropic work, she sought to interest municipalities in her plans, but the first approaches evoked no response; consequently, the first centre set up in Richmond at the Boorondara Kindergarten was financed by private subscription. So successful was the work that Richmond Council granted a subsidy, and centres soon followed at Carlton, Collingwood, Fitzroy and Geelong. In 1918 the committees of these centres banded together to form the Victorian Baby Health Centres Association, and this Association set up a non-residential training school for nurses in 1920 at South Melbourne, the first infant welfare training school in the State. In a report made in 1917 Dr. Younger directed attention to the assistance given by municipalities and sought government aid for maintenance on a £1 for £1 basis. This was granted and made retrospective. Thus were laid the foundations of an infant welfare service which was to become State-wide and to prove of incalculable value to Victoria. In 1916 Dr. Younger was married to Mr. John Ross. Their only child, a son, lost his life in New Guinea in the last war. Throughout her life Dr. Younger-Ross maintained an unceasing interest in maternal and child health work, and at the time of her death was vice-president and an active member of the Association she had pioneered. She was always ready to attend meetings and to spread the knowledge of the importance to mothers of preventive health teaching. This teaching, begun in Richmond in 1917, played no inconsiderable part in diminishing Victoria's infant death rate from 76·2 in 1917 to 18·37 in 1955. Mothers have so valued the help thus provided that today there are 531 centres in the State, and only three municipalities do not participate in the service.

Although many have played their part in pioneering the State's infant welfare service, its inception was the work of the late Dr. Younger-Ross, and those who knew her realize how wholeheartedly she dedicated her life to this work.

ERIC HAMILTON WHITE.

DR. W. J. MCCRISTAL has sent the following appreciation of the late Dr. Eric Hamilton White.

Eric White died at Kobe, Japan, on February 29, 1956, when on vacation as ship's surgeon. Though I was a privileged and intimate friend of Eric and his family, it was not my good fortune to see him other than at rare intervals, and I am indebted accordingly to his friends, Dr. W. H. Ward, Dr. W. W. Gunther and Dr. Alwyn Kinna for the following account of him.

After graduation in 1918, Eric White was resident medical officer at Sydney, Coast and Randwick Military Hospitals. Subsequently he served in Queensland in the hookworm eradication campaign, a subject on which he contributed to the literature.

In 1921 he married Miss Elsie Davis and commenced his work at Candelo, where he remained till he entered practice in Newcastle, succeeding Dr. J. B. F. McKenzie in 1936. Shortly after his arrival in Newcastle he was appointed an honorary physician at the Mater Misericordiæ Hospital and consulting medical officer to the Hillcrest Salvation Army Nursing Hospital. To both those institutions he gave loyal, devoted and unremitting service. In the field of public welfare work he was a foundation member of the Hamilton Rotary Club, whilst his spare moments were devoted to many fields of sporting endeavour, as a member of the Newcastle Golf Club, the Newcastle City Bowling Club and honorary medical officer of the Newcastle Trotting Association, and as a keen, knowledgeable and modest investor of the turf.

During those years he made a host of friends, who were attracted by his unobtrusive manner, his natural kindliness and his quiet, but delightful, sense of humour. His devotion to his family life was well known to all, and there was no prouder father than Eric in the thought that his three sons, Dr. Kevin, Dr. John and Dr. Geoffrey White, and his two daughters, Judy and Dorothy Anne, had followed his footsteps in the medical and nursing professions.

A memorial service, with the church packed by patients, friends and medical colleagues, and a moving valedictory address by Mr. D. Cannington, was a token of the affection in which he was held by all classes of the community and evidenced the great loss that will be felt by the citizens of Newcastle in his passing.

The following account has been received from Dr. John Broughton.

It was a rare privilege to have known Eric White for more than eight years. Beloved by patient, friend and colleague, he exemplified all the virtues which senior men demand in the ideal general practitioner; wisdom, humility, courage, equanimity, humour were all there, and above all a twinkling-eyed kindliness which, allied with tolerance, saw some good in the meanest of his fellows.

In his quiet, humble and industrious way Eric White "adorn'd the page" of general practice in this area, and his death has left his many friends greatly saddened.

"How wise must one be to be always kind." (Marie Ebner Eschenback.)

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 19, of April 26, 1956.

AUSTRALIAN MILITARY FORCES.

Citizen Military Forces.

Eastern Command.

Royal Australian Army Medical Corps (Medical).—To be Captain (provisionally), 13th March, 1956: 2/56844 Richard James McArthur.

Southern Command.

Royal Australian Army Medical Corps (Medical).—The provisional rank of 3/101831 Captain C. W. Baird is confirmed. 3/114023 Major C. A. Jones is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Southern Command), 15th February, 1956.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

Eastern Command.—The resignations of the following officers of their commissions are accepted: Honorary Captains H. L. Thompson, 5th January, 1956, and K. J. Trevillian, 14th January, 1956.

Southern Command.—The resignation of Honorary Captain A. M. Cuthbertson of his commission is accepted, 10th February, 1956.

The following officers are placed upon the Retired List (Northern Command) with permission to retain their rank and wear the prescribed uniform, 5th January, 1956: Lieutenant-Colonel (Honorary Colonel) D. A. Cameron, O.B.E., Lieutenant-Colonel B. F. R. Stafford, Majors (Honorary Lieutenant-Colonels) G. A. McLean and O. E. Nothling, Majors A. F. Foote and G. S. Hayes, Captains W. E. Hasker, I. G. Hooper, L. T. Jobbins and S. E. Warriner.

The following officers are placed upon the Retired List (Northern Command) with permission to retain their rank and wear the prescribed uniform, 17th February, 1956: Lieutenant-Colonel H. W. Johnson and Captain R. J. H. Spark. The following officers are retired: Honorary Major G. C. Taylor, Honorary Captains T. J. Cotter, K. J. Hill, V. T. J. Lynch, D. W. Neville and C. T. Underwood, 5th January, 1956, and Honorary Captains A. W. Chalmers and J. J. McIntosh, 17th February, 1956.

Royal Australasian College of Surgeons.

FINAL FELLOWSHIP EXAMINATION.

A MEETING of the Court of Examiners for the final examination for Fellowship of the Royal Australasian College of Surgeons in general surgery will be held in Sydney, beginning on Friday, October 19, 1956.

Candidates who desire to present themselves at this examination should apply on the prescribed form to the Censor-in-Chief for permission to do so before September 6, 1956. The appropriate forms are available from the Secretary, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1.

Candidates who have already been approved by the Censor-in-Chief but who have not yet presented for the examination, may present at this examination provided they notify the secretary of their intention to do so by September 6, 1956.

The examination fee is £21, *plus* exchange on cheques drawn on banks outside Melbourne, and must be paid to the secretary by September 6, 1956.

It is emphasized that entries close on this date and that late entries cannot be accepted.

The examination will be conducted in general surgery only.

FACULTY OF ANÆSTHETISTS.

Primary Examination for the F.F.A., R.A.C.S.

A PRIMARY EXAMINATION in anatomy, physiology, pharmacology and pathology for Fellowship of the Faculty of Anæsthetists, Royal Australasian College of Surgeons, will be conducted in Melbourne in March, 1957.

The written papers will be held on March 7 and 8, 1957.

The examination is open to graduates of not less than one year's standing of an approved medical school.

Candidates must submit evidence of their qualification and of the date of acquirement thereof.

Forms of application for admission to the examination may be obtained from the Secretary, Faculty of Anæsthetists, Royal Australasian College of Surgeons, Spring Street, Melbourne.

The fee for admission or readmission to the examination is £15 15s. (*plus* exchange on cheques drawn on banks outside Melbourne). The fee must be forwarded with the form of application so as to reach the secretary at his office not later than January 25, 1957.

It is stressed that entries close at the Faculty Office in Melbourne on January 25, 1957, and that late entries cannot be accepted.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following additions and amendments have been made to the Register of Medical Practitioners for New South Wales in accordance with the *Medical Practitioners Act*, 1938-1955:

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MAY 12, 1956.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	2(2)	2(1)	6(2)	1	1(1)	..	..	..	12
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	6	..	5(1)	..	..	..	..	..	11
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	1	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	1(1)	..	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	6(5)	6(6)	..	..	2(2)	..	..	..	14
Diphtheria	2(2)	11(11)	1(1)	..	4(4)	1	..	..	19
Dysentery (Bacillary)	..	3(2)	4(3)	3(3)	3	..	..	..	13
Encephalitis	..	..	..	1(1)	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	76(38)	50(27)	..	9(5)	3	6(3)	..	4	148
Lead Poisoning	..	..	2	..	..	..	..	..	2
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	4	..	..	..	..	..	4
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	5	2(1)	..	..	..	..	..	..	7
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	1	..	..	..	1
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	15(7)	5(2)	3(3)	2(1)	1	1	..	..	27
Puerperal Fever	..	..	2	..	..	..	..	..	2
Rubella	..	8(5)	..	1	..	..	..	..	9
Salmonella Infection	..	..	..	1(1)	2(2)	..	..	..	3
Scarlet Fever	16(8)	23(14)	..	7(6)	2(1)	1(1)	..	2	51
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	10	..	..	..	10
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	50(26)	22(18)	2(1)	4(2)	10(7)	2(1)	..	..	90
Typhoid Fever	..	1(1)	..	..	1	..	..	..	2
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (*a*) of the Act: Lane, Godfrey Joseph, M.B., B.S., 1932 (Univ. Melbourne); Coats, Howard Presswood, M.B., B.S., 1945 (Univ. Melbourne).

Registered medical practitioners who have complied with Section 17 (3) and are registered under Section 17 (1) (*b*) of the Act: Robinson, Derek, M.B., Ch.B., 1952 (Victoria Univ., Manchester).

The following additional qualifications have been registered: Campbell, Charles Haxton, M.R.A.C.P., 1954; Beirman, Basil Nathan, M.C.R.A., 1952, D.D.R. (Univ. Melbourne), 1953; Kerridge, Gordon, F.R.A.C.S., 1956; McBride, William Griffith, M.R.C.O.G., 1954; Sharland, Andrew Alexander, M.C.R.A., 1949, D.R., 1946 (Univ. Sydney).

TASMANIA.

THE following have been registered pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners: Holden, Dulcie Elizabeth, M.R.C.S., L.R.C.P. (London), 1944; Holden, Herbert, M.R.C.S., L.R.C.P. (London), 1943, M.B., Ch.B., 1943 (Univ. Liverpool); O'Reilly, Cyril Francis, M.B., B.Ch., 1936 (Univ. Belfast); Farrar, Derek Adrian Trickett, M.B., B.S., 1943 (Univ. London), D.L.O., 1955, F.R.C.S. (England), 1949.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Annual Subscription Course.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that Sir Lionel Whitby, C.V.O., M.C., M.A., M.D., F.R.C.P., D.P.H., Regius Professor of Physic, University of Cambridge, and Sims Commonwealth Travelling Professor for 1956, will lecture on "Anæmia in General Practice" on Friday, June 29, 1956, at 8.15 p.m., in the Stawell Hall, 145 Macquarie Street, Sydney.

This lecture will be given under the joint auspices of The Royal Australasian College of Physicians and the Post-Graduate Committee.

Congresses.

INTERNATIONAL CONGRESS OF GYNÆCOLOGISTS AND OBSTETRICIANS.

THE first Congress of *l'Union Professionelle Internationale des Gynécologues et Obstétriciens* will be held at Madrid on September 28 and 29, 1956. Further information may be obtained from the secretary-general, Dr. J. Courtois, 1, Rue Racine, St-Germain-en-Laye (S.-&-O.), France.

Medical Appointments.

Dr. K. J. Shellshear has been appointed Government Medical Officer at Beenleigh, Queensland.

Dr. G. C. Young has been appointed Superintendent of the Mental Hospital, Ararat, Victoria.

Dr. R. E. Davies has been appointed Public Vaccinator for the Shire of Deakin, Victoria.

Pursuant to the provisions of the *Quarantine Act*, 1908-1950, the appointments of Dr. William Hutchinson and Dr. Rex Richard Ashby Brock as Quarantine Officers, Northern Territory, have been cancelled, and Dr. Ronald Campbell Webb has been appointed Quarantine Officer, Northern Territory.

Dr. Bertram James Phillips has been appointed to the School Medical Service in the Department of Public Health, New South Wales.

Dr. Phyllis Edna Rodriguez has been appointed Registrar to the Radiotherapy Department at the Royal Adelaide Hospital, South Australia.

Dr. G. C. Young has been appointed Psychiatrist Superintendent, Mental Hygiene Branch, Department of Health, Victoria.

Deaths.

THE following death has been announced:

PALMER.—Henry Wilfred Palmer, on May 22, 1956, at Strathfield, New South Wales.

Diary for the Month.

JUNE 5.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 5.—Victorian Branch, B.M.A.: Branch Meeting.
JUNE 6.—Western Australian Branch, B.M.A.: Branch Council.
JUNE 8.—Tasmanian Branch, B.M.A.: Branch Council.
JUNE 8.—Queensland Branch, B.M.A.: Council Meeting.
JUNE 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 18.—Victorian Branch, B.M.A.: Finance Subcommittee.
JUNE 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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